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METHODS OF ALLERGIC STUDY

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The diagnosis of the etiology of asthma presents one of the most difficult problems in medicine. Making an allergic study is not merely making skin tests. It is necessary to attack the problem with all the means at our disposal, not using one method of approach only, since no single method is consistently reliable.

It is generally agreed that atopy is quite frequently present even though it can be demonstrated neither by scratch nor intracutaneous tests. Milk, for example, may give no cutaneous reaction although the ingestion of milk is followed by a prompt attack of asthma or allergic rhinitis, and the patient may assure one that milk always causes that reaction. Such an example found recently may be cited. A man, with asthma for about one year, on whom an allergic study is being made, had no cutaneous reaction to milk by the scratch and intracutaneous tests. For reasons not important here, an elimination diet was not used, but a diet was prescribed which varied from his usual one only in that all milk products were eliminated. Not until twelve days later did any change occur. At this time, he became asthma free. On the sixteenth day, after having no symptoms for four days, milk was ordered again, and within thirty minutes after drinking one glass of it, a marked attack of asthma resulted, necessitating epinephrin. Another attack followed in six hours. With the discontinuance of milk, this patient has had no more asthma. This food was suspected because his asthma had been aggravated after taking it for one month. Milk had been taken previously only in meager quantities; that is, only as it was used in cooking.

Conversely, positive skin tests, either intracutaneous or scratch, do not necessarily mean a clinical sensitivity. It is absolutely unjustifiable to assume that a case is one of bacterial asthma just because the skin tests are negative to foods and inhalants. It is first necessary to prove that foods and inhalants are not factors by elimination-diet trials and a sojourn in an air-filtered room. Elimination diets were devised to diagnose the skin-insensitive case, and they have proved to be a very valuable adjunct to allergic study. In fact, no study is complete without their use.

Useful diets have been published by numerous allergists, Rowe[1] being the pioneer in this field. These diets are well balanced, containing the proper amounts of protein, starch, fat, vitamines, and minerals. They particularly eliminate wheat, eggs, milk, potatoes, and orange juice—probably the commonest allergens from a food standpoint. Usually several different diets are used. Frequently, it requires fourteen days to be freed from the effect of an ingested allergen. Cooperative patients can easily maintain, and even gain, weight on any of these diets. If a patient can be made free from asthma on one particular diet, then foods can be tried out, one at a time, and each food tested for clinical sensitization separately. However, elimination diets have the disadvantage of including foods which are not infrequently a source of trouble, and in cases of multiple sensitizations to foods, all of these diets may include foods to which the patient is sensitive. It is desirable in certain cases to give a simple food exclusively, such as chemically pure dextrin or glucose. White

dextrin has the advantage of being more palatable than dextrose. One quart of a 10 per cent solution of either contains approximately 400 calories, and two or three quarts daily can be taken with fair ease. This type of elimination diet is useful in order to obtain information as to whether or not foods are a factor in the persistence of allergic symptoms. Its disadvantage is that it can be used only for a short time. Dextrin, exclusively, is particularly useful in relieving an acute attack of asthma. Once relieved by this diet, foods can be tested clinically, one at a time. Many observers have pointed out the advantage of dextrose therapy in the relief of asthma. It is interesting to note that epinephrin increases blood sugar.

As a group, skin tests for foods are less reliable than tests for epidermal and pollen antigens. If skin tests for these latter groups of substances are negative, it is necessary to confirm this by another method, namely, the allergen-free room. The room and each article of furniture must be thoroughly washed, to eliminate molds in the house dust; the mattress must be made of long staple cotton, because cotton sensitizations are not common. The offending substance of cotton is very uncommonly the long white fiber of the cotton seed. The brown short fibers nearest the seed are usually the offending substances. Another method used is to equip the room with a rubber mattress, rubber sheets and pillows. This room must have an efficient air filter and maintain a low positive pressure continuously. If no improvement results from remaining in this room for one week, it is probable that an air-borne antigen is not responsible for the symptoms.

Repeatedly observers have found that pollens are responsible for asthma and allergic rhinitis in spite of negative skin tests. Ophthalmic tests are useful in this event. Many allergists in certain cases advocate protecting the patient with the prevailing pollens whether they react or not. It is frequently found that reactions are obtained in these instances, when a fairly large dose is reached. This method should always be used if symptoms are aggravated during the pollinating season.

No allergic study is complete without investigating the bacterial phase of allergy. Most allergists recognize allergy due to bacteria, although there is considerable disagreement as to the method of diagnosing this condition. Bacterial allergy is not very common in childhood. It is more frequent later in life and, also, as the duration of asthma increases. It can be said that bacterial allergy is usually a secondary rather than a primary sensitization. Frequently, patients with hay-fever or asthma begin by having symptoms from pollens. Later these pollen symptoms continue throughout the year, the patients having become sensitive to their own organisms or to foods as well.

I believe overlooking the possibility of bacterial sensitization is the most frequent cause of failure in the treatment of allergy. Because a person is sensitive to foods and inhalants, does not preclude the presence of bacterial sensitization, which may be the most important cause of symptoms. A bacterial study must be made in every case unless prompt and entire relief is obtained by other methods. Bacterial allergy should be suspected if asthma is aggravated by respiratory infections, if asthmatic and nasal symptoms are worse in the winter, if the sputum is more purulent than mucoid, and if there are accompanying symptoms of a nasal allergy. Skin tests with stock bacterial antigens were at first advocated and later rejected by the majority of allergists. Later, autogenous vaccines, without skin testing with the individual organisms, were made and given with variable success. Later, all the individual organisms were isolated and the patient was tested intracutaneously[3]. Benson's[3] method is to culture the nasal secretion, sputum, stool, or other sources of infection—as teeth, tonsils, or sinus discharge, using blood agar and brain broth, isolating the different organisms and making individual vaccines of washed bacteria. filtrates of brain broth, or cultures of both. Then, test the patient with his own organisms intracutaneously. A minute dose, .01 cc., is used. It is best to use a standardized 1 per cent suspension of bacteria or an equal amount of filtrate, for testing. A positive reaction is assumed if an area of redness and swelling results within 24 to 48 hours. This reaction must be at least 1 cm. in diameter. A prompt wheal reaction is disregarded as probably nonspecific in the majority of cases, although there are exceptions in a few cases.

A great deal of controversy has arisen as to whether the delayed reaction is a non-specific one. Certainly, not all cases give it. Those maintaining that this reaction is non-specific, contend that if the same bacteria that gave a positive reaction are given to a normal individual, a similar reaction will result. This contention has been found to be true to a certain extent. Usually the reactions that occur in a normal person are not so pronounced as in the patient whose organisms were used, and they do not occur consistently. Very frequently a small dose of this bacterial antigen will provoke a sharp attack of asthma in the patient from whom it was cultured. Also, administering it in gradually increasing doses often relieves the symptoms. This latter phenomenon is evidence for the specificity of autogenous bacterial antigens. Frequently treatment doses have to be reduced to one-thousandth of a cubic centimeter (.001 cc.) to avoid excessive reactions. It is interesting to observe that bacterial allergy appears to be quite different from atopy. The former usually produces no wheal and reagins are usually not demonstrated by the Prausnitz-Kustner or passive transfer reactions. True atopy produces blood-serum reagins and usually produces a wheal reaction on the skin.

In proceeding with an allergic study, a very carefully detailed history is most important, frequently giving valuable diagnostic information as to etiology. Particular attention is to be paid to the nose, looking for allergic rhinitis and especially polyps. Smears of sputum and nasal secretion must be made and stained with the routine blood stain to determine the percentage of eosinophiles. It is generally conceded that nasal polyps and abnormal percentages of nasal and sputum eosniophiles are pathognomonic of allergy. Abnormal amounts of blood eosinophiles are suggestive of allergy.

Sinusitis is usually a secondary infection and not a primary cause of allergic symptoms. An allergic nose is a fertile field for the development of a sinus infection. Relieving a cavity of blocked secretions can conceivably relieve allergic symptoms if bacteria to which the patient is sensitive are eliminated. Scratch tests of the skin are performed preliminary to intracutaneous tests, if the former have not previouly been given and found negative. This is done to avoid the danger of a general reaction from the injection of many antigens at one time.

The following is a good routine procedure for allergic study:

1. A very careful thorough history.
2. A complete general examination, including x-ray of the chest.
3. Examination of the nose and sinuses, including x-ray and transillumination as indicated.
4. Examination of blood, urine, stool, sputum, and nasal secretion. Other procedures as indicated.
5. Scratch tests for foods, epidermals, and pollens.
6. Intracutaneous tests for foods, epidermals, and pollens.
7. Bacterial study of sputum, stool, nasal secretion, and intracutaneous testing of the individual organinsms.
8. A week's stay in an allergen-free room.
9. A thorough trial of elimination diets.
10. Passive transfer tests on certain cases.

The results of the above procedure must be correlated with the history, and a plan of treatment can then be based upon all of the information gained. Often, it is advantageous first to desensitize to certain substances before other procedures are tried.

In conclusion, we may say that it is only by using all of the methods of allergic study which we now have at our disposal that we can obtain correct diagnoses of asthma and, consequently, a higher percentage of success in this, one of the most baffling problems of clinical medicine.

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DISCUSSION

DR. L. O. DUTTON (El Paso): I wish to commend Dr. Kibler for making so clear the fact that an allergic study must be exhaustive and complete to insure success in uncovering the offending factors in asthma and allied conditions. His outline is complete and serves to indicate the exhaustive means necessary in some cases to arrive at a diagnosis as to offending factors.

I wish to take this opportunity to offer my discussion of all the papers presented on this subject.

In the treatment of hay-fever we prefer the preseasonal or perennial course, but very satisfactory results can be obtained by proper coseasonal desensitization.

Of sixty cases, forty-three were treated by coseasonal injections, with 100 per cent relief in twenty-five cases; ninety per cent relief in nine cases; eighty per cent relief in three cases; seventy-five per cent relief in three cases; fifty per cent relief in two cases; complete failure in one case. Seventeen cases were treated preseasonally with 100 per cent relief in fifteen cases, ninety per cent in one case and seventy-five per cent in one case.

Failure is most often due to improper dosage or to unrecognized secondary factors. The dose of extract necessary to desensitize varies within very wide limits and it is very necessary to avoid both under and over dosage.

Secondary factors are also important. In the sixty cases outlined above, there were twenty-two cases which failed to receive the maximum benefit until the secondary agents (foods and dusts) were recognized and eliminated.

In the control of asthma, our greatest stumbling block has been non-cooperation of the patient. Those who have faithfully carried out our directions have almost always eventually been relieved. The factors in asthma are so diverse that it frequently requires considerable time to arrive at a final diagnosis of the complete cause. We find it of value to spend considerable time in educating the patient as to the reasons for the various procedures employed.

The benefit to be derived from food-allergy studies in a variety of conditions is illustrated by ninety-five cases. There were sixty-three excellent results, nineteen fair results, and thirteen failures. The conditions occurring in the list were gastro-intestinal upsets, eczema, headache, angina pectoris, urticaria, colitis, acneform eruptions, arthritis, frequency of urination, stomatitis, sinus congestion, and nervousness.

We feel that many patients who could be greatly benefited are not given the opportunity to be adequately diagnosed from the standpoint of allergy.

DR. LESLIE M. SMITH (El Paso): While this essay was concerned more with the non-dermatological manifestations of allergy, it might not be out of place here to consider the different methods of allergic study in relation to the several groups of skin diseases with which allergy is more or less associated, and to demonstrate by lantern slides the clinical appearances of these conditions.

Urticaria and the allied neurovascular disturbance, angioneurotic edema, are well known to be frequently caused by food allergy. They are also caused occasionally by bacterial allergy. The method of study here is, of course, the scratch and intracutaneous tests. Not infrequently these tests will be negative. In these cases the conditions may be due to some physical agent or to some toxic product of disordered digestion rather than to a specific food allergen. Of the so-called eczematoid eruptions, few of them are found definitely due to the common allergens for which tests are usually made.

The condition known as neurodermatitis should not be considered an eczema. These cases are prone to develop in allergic children and are quite persistent. Some of them are found sensitive to foods and other allergens by the scratch or intradermal method. In my experience, however, there is usually also a nervous instability. I have repeatedly seen these cases flare up after worry or other emotional excitement; I have seen none entirely cured by removal of offending foods or other allergens.

The type of dermatitis which dermatologists today call eczema, is more likely to be associated with external irritants with which the patient comes in contact, such as dyes, various chemicals, furs, silk, wool, insecticides, and medicaments such as picric acid, as demonstrated on one of the slides shown. In these cases patch tests based on suspicion obtained from the history and with proper dilutions often yield valuable information. The condition known as infantile eczema is often due to specific food sensitizations.

SOME HAY-FEVER PROBLEMS IN THE SOUTHWEST

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(Read before the nineteenth annual session of The Medical and Surgical Association of the Southwest, held at El Paso, Texas, Dec. 7 to 9, 1933.)

Our peculiar geographical and climatological situation in the southwest begets a problem in seasonal hay-fever that is more or less unique. Several factors enter into this: the marked variations in altitude, and therefore equally great variations in length and intensity of warm and cold seasons, in quantity of rainfall per annum, the directions, frequency and velocities of our winds, the types of soil—all tend to produce a flora that is somewhat distinctive of this geographical area. We may find some important pollen-bearing plant in abundance in and around El Paso, for example, and this particular plant may be of secondary importance or even absent in Tucson or Phoenix. All these, and other factors, tend to complicate the successful diagnosis and treatment of pollenosis in our communities, but the fundamental problems are the same here as elsewhere.

There is one problem that I shall mention in passing—it is mentioned only as an unsolved problem. I have not been able to find the answer in literature, but I shall attempt to show that it deserves serious consideration. Many of our localities are extremely arid, the atmosphere is dry and often very hot, the humidity is low; can or does this very dry air, per se, affect the nasal and bronchial mucous membranes of some patients, rendering them more likely to acquire a state of hypersensitivity than they would in a different cilmate? It is not infrequent to encounter a patient who has not the typical signs and symptoms of a hypersensitive disease, but who complains of a distress in the nose which has developed after coming to this climate, and which is characterized by a dry crusting, slightly pale, nonedematous mucous membrane, and is relieved by application of some bland oil or ointment. Is such a nose more apt to develop hay-fever than it would in a more humid region? I suspect that it is.

A further distinctive characteristic of this climate with reference to seasonal pollen disease is the well-known fact that the pollen, which is to be an important factor in the causation of this disease, must be wind-borne. Relatively we have few such plants, but those we have are profuse pollinators, and the paucity of rainfall, low humidity, and the relatively high temperatures of summer encourage this characteristic. This was very definitely and graphically shown by Marjorie Hopkins' in 1931 when she kept a close record in 103 cases of ragweed hay-fever, correlating the symptoms manifested by these patients with the temperature, humidity, and pollen counts, daily. It was noticed that the humidity curve was inversely proportional to the symptom curve and the pollen curve. The rainfall also tended to lower the pollen curve, and thus lowered the symptom curve. It was also noted that, when the temperature was high, the pollen curve and the symptom curve were usually high. She emphasized the fact that the variations in humidity and temperature were considered as affecting the symptoms of the patient, not directly, but through their influence on the pollen production. These observations are borne out in our own section. We have the high temperatures, the low humidity, and abundant pollen production therefore, an ideal condition for aggravating any already existing pollenosis and for producing grave symptoms in those patients who have the least degree of inherent hypersensitivity.

There is an idea prevalent among the laity, and often poorly understood by the profession, that the dust off the desert and unpaved streets, and our not infrequent windstorms that carry huge quantities of dust into the air, aggravate hay-fever symptoms; and that they are often the actual cause of the symptoms. It has long since been my personal opinion that the dust and sand-laden air which is inspired could act as a non-specific irritant to the respiratory mucous membranes and aggravate an already existing hay-fever, but by far the greatest irritant is the unseen pollen granules carried along with the dust and sand. Street dust, in and of itself, probably never caused hay-fever, but it could act as a nonspecific irritant to aggravate the damage done by pollen. It is so rare as to be almost unknown to have an exacerbation of seasonal hay-fever in winter, notwithstanding equal amounts of atmospheric dust in summer and winter. This fact makes the following case more difficult to explain. W. C., a known Bermuda-grass hay-fever patient, was in Central America. He had occasion to walk considerable distance through a lava bed. There was no apparent vegetation for miles around, lava covered the ground completely. Walking in this lava caused a fine white dust to fill the air. Breathing this precipitated a violent attack of hay-fever. Apparently there was no possible inhalant save the dust from the lava.

We have shown that we have more pollen in the air for longer periods of time than places with cooler and more humid climate; therefore, we have a greater exposure for longer consecutive periods. We have the drying effect on the mucous membranes, with its consequent sensitizing effect; we have the lesser important irritating factor of the dust and sand. These conditions tend to increase the number of victims of this malady; they cause patients to develop it here who never had it before coming here and who probably would not have had it, had they not come to a dry climate, and aggravate those who have the inherited tendency or who already have hay-fever. We are all familiar with the patient who, having terrific hay-fever, goes to a cooler and damper

region, and is immediately relieved until he returns here. Is this because he has gotten away from the plant or plants to which he is sensitive? Perhaps so, in many cases, but this can not apply to those patients who are sensitive only to one pollen, Bermuda grass, for example. They usually experience the same degree of relief by a sojourn in a community where there is more rain and dampness, although Bermuda may be more prevalent than it is here. I believe the explanation of this phenomenon is threefold: the difference in degree of exposure, which in turn depends upon length of seasons, amount of pollen washed from air by rain or fog, and so forth, the difference in humidity which affects the drying of nasal mucous membranes, and the difference in the quantity of atmospheric dust as an additional irritant.

The monumental work of Piness and Miller[5] bears out this belief to an extent. They studied two entire communities which were in many ways comparable to our own here. One was a potash-mining center with much alkali dust in the air; the other was a town built around a smelter. The high incidence of hay-fever in these places led to the belief that fumes from the smelter and dust of the mines were the important etiological factors. Piness and Miller made a botanical survey of the area, tested the patients with appropriate pollens, found the cause to be wind-borne pollens, and had the usual success by the treatments with pollen extracts. They concluded that, by the excess exposure to pollens, symptoms were caused in greater percentage of population than is usually shown in statistics. Neither the fumes nor the dust caused the symptoms. It is my contention that these people get some non-specific irritation of their mucous membranes from the hot, dry air and the dust, which renders them more susceptible to becoming sensitized; this type of irritation, plus the excessive exposure to hay-fever-producing pollens, aggravate the condition and make successful treatment more difficult.

The work done by other observers has left little doubt of the hereditary character of allergy. The study of Bray[3] in London has done much to make this clear. It is worthy of note that we have a lower positive family history of allergy in our hay-fever cases than the average we find in studies elsewhere. In 344 cases of hay-fever, Rackemann[4] found 35 per cent to have positive family history; Balyeat[6] reports 60.1 per cent positive in hay-fever and asthma; Kahn[6] had 80 per cent in 100 cases of hay-fever; Spain and Cooke[7] obtained positive hereditary history in 58.4 per cent of 462 hay-fever patients. In our own series, we found 27.6 per cent had a positive family history of some allergic manifestations. Since this percentage is lower than the average of authors quoted above, we feel that the climatological factors, with the drying effect on the nose and abundance of pollen, cause symptoms to occur in many of our patients who have so little hereditary hypersensitivity that the symptoms would not become manifest where the patient did not encounter the aggravating side effects of the natural forces.

As stated above, due to differences in altitudes, length and intensity of seasons, variations of rainfall in different sections, and amount of irrigation, the flora of one district of the southwest will differ greatly from another, although not separated by many miles. For instance, I understand that Russian thistle (Salsola pestifer) is considered an important factor in El Paso; in the vicinity of Tucson, it is so rare that it is quite unimportant; thus it is throughout our particular geographical location.

It is not within the province of this paper to discuss in detail the important pollen-bearing plants in any one community. Suffice it to say, that in Tucson we consider Bermuda grass (Cynodon dactylon) the most important of all; it comes on by April and continues to pollinate until November or early December. In the fall, August 15th to November, there are two others almost as important: slender ragweed (Franseria tenuifolia) and careless weed (Amaranthus Palmeri); the salt bush or shad scale (Atriplex canescens) is almost equally as important. A host of others could be mentioned, time permitting, but none equal to these in their hay-fever-causing proclivities.

Under the subject of adequate treatment, I propose to mention several points that I consider worthy of thought. First, may the time soon arrive when we will all speak the same language in the matter of dosage; be it pollen units, nitrogen content, percentage solution, A-B-C-D dilutions, or what not: uniformity

will clarify our study of the literature immensely.

As we read the literature, we are at once impressed with the great variation in what the different authors consider the optimum dosage of pollen extracts.

Grafton Tyler Brown[8] uses as maximum dose 1 cc. of 6 to 10 per cent solution; J. Mercer Anderson[9] advocates the use of small doses, none to exceed 0.15 cc. of 1:100 (1 per cent) and most 0.1 cc. of 1:500 or less. Thus, we have two extremes. It seems to me that the optimum dose should be the minimum amount of pollen extract that is compatible with protection of the patient against symptoms. A case in point:

Mrs. M. was treated elsewhere with Bermuda-grass extract. When she attained a certain dose, the size of which I do not know, she had an untoward local reaction and constitutional reaction consisting of marked increase in hay-fever symptoms and asthma. From that time on, each subsequent dose was apparently increased and gave similar or worse reactions. I treated her this season with complete relief of symptoms; when I gave her 0.3 cc. of 1:1000 solution, she had a large local reaction. I dropped to .2 of 1:1000 solution without any subsequent increase in dose, accompanied by a complete relief of hay-fever. I feel that we should attempt to classify our patients according to Cooke[10] et al as nearly as possible; then proceed without any preconceived ideas as to what the dosage is to be, but be guided by reactions and symptoms.

This suggests to us the question of preseasonal, coseasonal, or perennial treatment. We need only to read the current literature to see that the perennial treatment, according to Brown[11], Unger[12], and others is preferred by most authors. I am in accord with this conclusion. I do take exception, however, to the treatment in this section of the country by the preseasonal method. Our season is much too long, and the immunity produced is much too transient to hope to protect our patients with a brief preseasonal desensitization. This is another reason why I think the observers in the east have little conception of what our hay-fever problem is in the southwest.

One of my greatest antipathies is the so-called prepared treatment set that is put on the market by the various manufacturing concerns. They should be condemned because, when the usual series of fifteen injections are completed, the patient, and perhaps the doctor, thinks he has finished the desensitization and that he has had all that is necessary to protect him through the season. Most often this is hopelessly inadequate. It gets the doctor and the method of treatment in ill repute. It should further be condemned because of the inflexibility of the doses. It is often necessary to repeat the former dose or even give less than the preceding, and this cannot be done with the serially dosed preparations supplied by so many manufacturers. The use of these prepared sets is bound to lead to disappointment and failures in obtaining satisfactory results.

Believing that preseasonal treatment is inadequate, and the perennial treatment is decidedly the most efficacious, we are still forced to treat practically all cases coseasonally, with all the attendant dangers, making extreme care even more imperative than we are taught by the authorities. The intervals between doses and the percentage increase in the doses, must be considered most carefully. It is our custom to begin the treatment with three injections weekly, beginning with 1:100,000 to 1:10,000, depending upon tolerance. As time goes on, and a larger dose is attained, this is changed to twice weekly, and finally once weekly when we have reached what we consider the maximum dose. This is continued throughout the season unless symptoms recur; then we find that if injections are given more often, usually symptoms immediately decrease. In giving treatments as often as three times a week, we do not increase the dose so rapidly as some—often .05 cc. or less, depending, of course, upon reactions. We feel, like Vaughan[13], that if the treatment gives 75 per cent of the patients less than 90 per cent relief, the blame is on the doctor, and the treatments are not adequate.

We are all familiar with the multiple sensitivity of most patients, and, as pointed out by Gelfand[14], many of our failures are due to sensitivity to allergens that are not pollens. However, inhalants are the greatest single cause, by far, and it is only in the rare case that success or failure in treatment is going to depend upon a food offender. Thus, in failures, we should study particularly the inhal-

ant group for the cause, more especially to see whether we are overlooking an important pollen.

No paper of this kind would be complete without mentioning constitutional reactions, the great "bugbear" of all desensitization methods. Waldbott[15] has pointed out the causes of practically all of them. I believe it can all be summed up in one answer, viz., overdose. Whether the overdose is due to too large a single dose, too frequent injections, an exposure to a different antigen in conjunction with the pollen extract, pollen absorbed through the nasal passages in addition to the extract administered, it invariably represents an overdose. In some 7,000 injections in the past three years, we have had five constitutional reactions; in none of them was there an unusual increase in dosage, nor a change from old to new extract. The explanation that seems most plausible is that the injection was not the only cause of the reaction, but the pollen absorbed by the mucous membrane was the contributing element that, in conjunction with the pollen extract, was enough to cause the reaction. These reactions were all similar in each of the five instances characterized by an aggravated attack of hay-fever: profuse watering of nose and eyes, protracted sneezing, swelling of lips, eyelids, tongue, oral, and pharyngeal mucous membranes, generalized urticaria; in one there was asthma. Four occurred in 10 to 20 minutes after injections; one some 4 or 5 hours following usual treatment. In none was a very concentrated solution used, the greatest was .1 cc. of 1:100 solution. All were readily controlled by the use of epinephrin. It has been our custom to drop back to about the fourth or fifth preceding dose and work up very slowly, always stopping below the dose which precipitated the constitutional reaction, and to date we have not had two to occur in the same patient.

Means of preventing reactions—such as the use of tourniquet as advocated by Duke, mixture of the extract with epinephrin before administration, the withdrawal of the syringe plunger to assure one that the needle is not in a vein, hard pressure with cotton sponge over site of injection to prevent back seepage into a vein, having the patient wait in the office to observe the local reaction, and so forth—are familiar to all. As important as they all are, they are insignificant in comparison to the selection of the proper dose, as constitutional reactions are always due to an overdose.

Finally, I wish to reiterate that failure to relieve more than 90 per cent of seasonal hay-fever symptoms is a reflection on the doctor, and when this occurs, he should go into consultation with himself and analyze the cases more closely to find wherein the causes for failure lie.

SUMMARY

1. Due to different natural forces in the southwest, we have a hay-fever problem here that is unique.

2. The low humidity, the hot dry air and possibly dust, and the winds, add their quota to the difficulties of successful diagnosis and treatment.

3. Importance of knowledge of local wind-pollinated flora, adequate treatment, proper dosage, and systemic reactions, are discussed.

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DISCUSSION

(Of Papers of Drs. Wilson and Smith)

DR. GEORGE TURNER, El Paso (opening):

Speaking of Dr. Smith's paper. It is extremely practical, dwelling on the practical side of treatment. Both papers touch on etiology. A most important point was brought out and that is that pollens differ in different localities, even in this arid country. Pollens that cause the greatest trouble in Phoenix are different from those in Tucson or El Paso. For example, ragweed has been almost unknown in El Paso, but within the last few years some western ragweed has begun to appear along the roadways. The most common pollens in this locality are (1) cottonwood, (2) Russian thistle, (3) Bermuda grass and (4) careless weed. Dr. Wilson mentioned climate as a possible cause of so many cases of hay-fever, on account of the drying effect on dust or pollen. This locality was once a lake bed, which might add some factors in the dust, such as alkali or iodine. Referring to the observation that pollens differ in different localities, the toxicity of the same pollen will vary according to the climatic conditions under which it grows. With regard to Bermuda cases who go back, before desensitization is completed, to where there is plenty of Bermuda but do not have hay-fever; it is possible that the atmosphere conditions have much to do with this or the type of pollen produced in the two places may be different. In the arid climates the pollens are more toxic. Hay-fever is a local problem; you should know the pollens of your own locality before treating patients. If the toxicity of the pollens is different, it will be impossible to standardize on any given quantity. In Dallas, Dr. Black standardizes his maximum dose on 1 cc. of 1:10 dilution. You take the western pollen and give 1 cc. of 1:10 dilution and you will get a strong reaction, no matter how much you have desensitized the patient. The dosage will depend on the toleration of the individual patient and must be just below the toleration dose.

With regard to treatment. To the important questions of seasonal or pre-seasonal treatment, another should be added, that is, the prevention from the public health standpoint. In these arid regions very few of the hay-fever producing plants are in the cities, and those present are brought in by irrigation. We are growing the plants that are directly responsible for the affliction from which we suffer. Several years ago, hay-fever was bad in El Paso, because much building and street work had dug up the ground and left it moist enough to grow Russian thistle. We had a crop of this every year and much careless weed. It is entirely possible to curb hay-fever by weed eradication and an active campaign was begun for weed destruction, with the result that the incidence of hay-fever in El Paso was decidedly decreased. Treatment is important but prevention is a great help in cutting down the amount of hay-fever. Year-round treatment is probably the most important; preseasonal treatment to the point of 1:100 dilution will not give a sufficiently lasting immunization to carry patients through a long season. If carried into the season, you may run into reactions by the addition of pollen to what is given by injection.

DR. CHAS. S. KIBLER (Tucson): Wish to congratulate Dr. Wilson on his paper on the peculiarities of the problems in the southwest. Plants do change their habitat and adaptability. Some twelve years ago we found no Russian thistle in Tucson, but it is now present and has increased to the point where it is a real problem. Dr. Phillips has called attention to the fact that the same thing has occurred in Phoenix, that is, Russian thistle coming down into the lower altitude. Careless weed, which is a profuse pollinator in the fall, is apparently adapting itself to higher altitudes. A most important factor in the southwest is the dryness of the atmosphere. One per cent over the country and four to five per cent of the population in Tucson have hay-fever. Patients who get complete relief at the coast do so because of the humidity. The best preservative of pollen is dryness; in the southwest, pollen produced in a given day is not destroyed by the moisture and finally settles in the dust, and is blown up in the air the next day or later. In relation to Dr. Smith's paper. In an off season when the patient has had the maximum dose, it is not necessary to inject oftener than once a month. This will afford adequate protection in an off season.

DR. E. W. PHILLIPS (Phoenix): Just a word about Russian thistle. It took six years to work into Phoenix and get so it could live. I predicted some years ago that it would get around to produce a fall crop. This year it did make a fall crop, a secondary growth from the stalks that dried up last June.

DR. HILGARTNER (Austin, Tex.): The hay-fever problem always attracts attention. In central Texas the chief offender is cedar, and citizens wanted to cut down all the cedar for many miles around. From the point of view of the rhinologist, the papers you have heard today and those seen in the literature do not sufficiently consider the conditions of the nose. In the eyes of the layman, any watering of the eyes is hay-fever. There are three types: (a) that due to spurs or polyps in the nose; (b) that due to hypermia or turgescence of the mucous membrane of the turbinates; (c) that due to plant pollens. In central Texas we have to contend with cedar, Bermuda grass and ragweed. We have many patients cured by removing polyps and spurs. The desensitization has been rather unsatisfactory in our hands and that of our associates in Austin. I would like to suggest two procedures that have been successful and not yet mentioned. The first is the method of Zinsky: this is the use of the diathermy current and just touching the mucous membrane of the turbinates with the positive electrode; this changes the lymphatic supply of the turbinate; with shrinking and desiccating the mucous membrane. The method that has been successful in the hands of Dr. Loving and myself in fifty or sixty cases is called the zinc

ionization. The nose is anesthetized by cocaine or some other anesthetic, and is packed with a one per cent solution of zinc sulphate on a tampon. Galvanic current is used with the electrode in the nose, 1 to 5 ma. and 15 to 20 minutes. The zinc salt splits up, with the deposit of metallic coating on the mucous membrane of the nose. The membrane shrinks down, the turbinate gets smaller and the patient gets along much more comfortable following this than where many injections have been taken. It is quite rare that as many as three treatments are necessary. From 75 to 80 per cent get complete relief or cures for two, or three years, which is as long as we have used the treatment. It is a procedure of choice and men working along hay-fever lines should consider it. Some use the treatment twice, first on one side and then on the other; it causes some discomfort, so they do not do both sides at one sitting. Once in a while you get asthma which can be taken care of with adrenalin or ephedrin.

DR. WILSON, Tucson (closing): I wish to thank the men for the discussions. Dr. Turner brought up some points worthy of consideration. In spite of what has been said about climate, Bermuda grass may not affect patients the same; it is a matter of difference in pollens. Do not care for the removal of polyps, spurs or surgery in the nose for the relief of seasonal pollen disease, though it may relieve what appears to be hay-fever to the patient. With regard to zinc ionization, want to know more about it. Recently, men in Philadelphia have worked with the application of pure phenol to the nasal mucous membrane; fifty or sixty cases have gotten relief through a whole season. This should be followed up.

REACTIONS IN BLOOD TRANSFUSIONS

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(Read before the nineteenth annual session of The Medical and Surgical Association of the Southwest, held at El Paso, Texas, Dec. 7 to 9, 1933.)

Blood transfusion has become the most common therapeutic agent at our disposal today. It is almost the universal treatment for every known ailment that we come in contact with. The diseases in which it is absolutely contra indicated are very few and obvious. In polycythemia, of course, we are dealing with too much blood, and in certain cardiac cases where a failing heart is already over-worked the strain of additional blood will often result in acute failure and death.

At present, blood transfusion has become so common a practice among practitioners, that a word of warning should be given against "familiarity breeding contempt.'"

Back in 1920, Dr. J. Pemberton[1,2], of the Mayo clinic, said in an article appearing in the Iowa State Journal of Medicine, "The operator should be conversant with all the dangers associated with the transfusion of blood. He should exercise judgment in advising the procedure and he should be conscientious in the selection of donors. The application of this valuable therapeutic measure must not be undertaken without a careful consideration of all the dangerous complications that may follow. The procedure is very often considered only a simple intravenous medication or a minor operation, while in reality its potential dangers place it with the major operations."

As early as 1667, Jean Baptiste Denys described in vivid style the outstanding features of the so-called hemolytic transfusion reactions, which until recently served as an inexplicable and insuperable barrier to the transfusion of blood. An understanding of the nature of the incompatibilities that are responsible for these frequently fatal reactions came only after the studies of Landsteiner on hemagglutination in 1901. A simple method for determining compatibility grew out of the classifications of Jansky (1907) and Moss (1910). As a result of these discoveries, transfusion of blood became a popular therapeutic procedure at about the time of the world war; today, provided one has taken the necessary precautions in preliminary grouping and cross-agglutination tests, it is a procedure relatively free from danger.[3] Nevertheless, transfusion reactions still occur, and although some of them are undoubtedly due to carelessness in the preliminary steps, it has been learned that certain reactions, most of them mild but others severe and fatal, can not be clearly explained on the basis of the obvious incompatibilities discovered by Jansky and Moss. In perusing the literature I have found that nearly every author attempts to classify these reactions. All of them are based primarily on symptomatology, and are designed for the guidance of the operator of the transfusion.

These reactions can be classified as hemolytic, chemical, and proteolytic. The proteolytic can be further subdivided into allergic and anaphylactic.

The reactions due to hemolysis and agglutination are always serious and often fatal. Those falling under this heading are by far the most severe. Death has been known to occur from the introduction of only 44 cc. of blood. In the majority of the cases the reactions manifest themselves rapidly, and if the patient is being watched very carefully by the operator, he can stop the procedure before a sufficient amount has been given to cause death.

The first symptom is usually subjective, the patient complaining of severe pain in the lumbar region of the back. This is quickly followed by flushing, and complaints of feeling hot all over the body, especially the chest and face. Following the hot flush there is paling of the skin, the face often becoming livid. Profuse perspiration, dyspnea, cyanosis, failing pulse, and dilatation of the pupils, are accompanying symptoms. The patient often becomes panicky and will call on the doctor to do something, because death seems imminent.

Usually if the transfusion is stopped promptly and adrenalin is administered by hypo in 15-minim doses every fifteen minutes for several doses, the patient will rally. A sharp chill and rise in temperature usually follows this type of reaction. Atropine 1/150 grain, combined with morphine, will be beneficial and promote the comfort of the patient.

These acute symptoms are usually followed in a few hours by hematuria, with the presence of albumin and casts in the urine, and sometimes by anuria, which always adds a serious complication to the situation. A blood examination will show evidence of acute hemolysis.

In those cases where death occurs, the reaction generally runs a peculiar and highly characteristic course which presents the following features: Immediately after transfusion, there is a sharp febrile reaction, followed frequently by hemoglobinuria and invariably by suppression of urine. There is an interval of several days during which there is symptomatic improvement but continued oliguria, and jaundice appears. After this interval the characteristic features of the delayed reaction develop rapidly. They usually begin with agitation or drowsiness which is replaced by out-spoken evidence of uremia; convulsions and coma may supervene. In those cases that recover from these severe reactions, the picture does not begin to change usually until about the twelfth day, complete recovery taking about three weeks.

Acute and massive blood destruction by hemolysis throws a tremendous load on the kidneys in the effort to clear the blood stream of its broken down hemoglobin, proteins, and cellular debris. These elements are insoluble in an acid medium and, consequently, the tubules of the kidneys become clogged, resulting in interference with normal function, which causes urinary suppression, edema, coma, and death.

These severe cases may be benefited by intravenous injections of glucose, alkalinization, and diathermy to the kidneys.

The real treatment, however, lies in the field of prevention. Since one fatal case which occurred in St. Mary's Hospital (Tucson) over a year ago, there have been no hemolytic reactions in twelve cases on my service. This case was seen by me in consultation three days after the transfusion. The cause of the error was never determined, because, in addition to the donor being in the same group as the recipient, the bloods were cross-typed. We believe the possible error lay in the fact that the cross-typing was observed only thirty minutes, which is not sufficient length of time to observe clumping or hemolysis in some slow-acting sera.

Since that time all cross-typing is observed for a full forty-five minute period, and under constant temperature. We use only the grouping of Jansky and Moss to determine what group the patient is in. All bloods are cross-typed, for every transfusion—fresh blood being used. Such a procedure is time consuming but we believe it is worth while.

We must include in this class of reactions, those occasionally seen after transfusion of individuals suffering from certain pathologic conditions in which there is a very active hemolytic agent at work. These are not constant but may appear in such conditions as pernicious anemia, purpura, hemolytic jaundice, leukemia, malignancies, and sepsis. This type of reaction can be neither foreseen nor avoided, but the possibility of its occurrence should be kept in mind so that prompt measures may be instituted to counteract its effect should it occur.

In speaking of the chemical reactions, I refer to those reactions occurring where certain

chemicals, such as sodium citrate and saline, are used with the blood from the donor. The symptoms are so similar to those of the hemolytic reactions that we can not differentiate them symptomatically, except that they are usually milder, and where whole blood is used between the same individuals the reactions do not occur.

I do not wish to enter into the controversy of citrated blood versus whole-blood transfusions; both sides have their good points. In isolated communities, with refractory children, and so forth, the citrate method unquestionably is the better procedure.

I use the direct method altogether. The apparatus is the Scannell, which, briefly, is a 20 cc. syringe with a three-way pet-cock attachment, whereby the blood is withdrawn into the syringe from the donor and is forcced into the recipient when the pet-cock is turned and the piston of the syringe pushed home. The third way is used to wash the syringe out with sodium citrate solution.

Professor Lawrence H. Snyder[4] of North Carolina State College, in his treatise on "Blood Grouping in Relation to Legal and Clinical Medicine," says: "There are both advantages and disadvantages to the citrate method. The disadvantages are the possible occurrence of citrate reactions, the danger that the availability and simplicity of the method may lead to the performance of the operation without sufficient experience, and the possible bad effects of citrate upon blood. Citrate reactions are known to occur. Various workers have found reactions occurring after citrate transfusions. There seems to be a variation in the tolerance of different people for sodium citrate. It has also been stated that citrate renders the corpuscles more fragile, diminishes complement in donor's serum, reduces the opsonic index in the plasma, reduces the phagocytic power of the leukocytes, and destroys platelets."

In my opinion, if a transfusion is really needed the most benefit is certainly to be derived from the whole unadulterated blood.

As I said before, I consider the hemolytic reactions, by far, the most important and severe; but there are many cases on record where the proteolytic type have been extremely dangerous, and a few fatalities have occurred.

Of the allergic reactions, two fairly distinctive types are seen: the mild febrile type and those showing urticarial wheals, asthma, and so forth. In checking over the literature, reports indicate that twenty per cent of the cases where the direct method is used, show the mild febrbile type. Approximately five per cent of these are accompanied by a chill. I could find no definite statistics on the indirect or citrate method.

Any rise in temperature of one degree or more above the average level before transfusion, and occurring any time within twenty-four hours following the transfusion, is considered a reaction. Those cases where the rise in temperature is accompanied by a chill show the most marked reactions.

These cases always cross-type perfectly, and there is never any indication in the urine or by any tests that hemolysis occurs. They always respond very readily to adrenalin chloride in 15-minim doses. As a prophylaxis one may use fasting donors on those cases that are known to react in this manner. It is not practical to use them, however, routinely.

About ten per cent of individuals show the other type of allergic reaction, where one gets the skin manifestation, intestinal upsets, or asthma. It varies in intensity from a slight erythema to general urticaria, at times involving all the mucous-membrane surfaces, with swelling of the eyes, lips, tongue, larynx and bronchi, as evidenced by respiratory difficulty, and even intestinal cramps and diarrhea. A chill and elevation of temperature may or may not accompany these symptoms. In its most severe form it can seriously endanger the life of a patient. The reaction may manifest itself after only a few cubic centimeters of blood has been introduced and usually is not delayed longer than twenty minutes after the transfusion is completed, but there are a few cases on record where the patient has developed urticaria several days after the transfusion. The prompt administration of adrenalin chloride will relieve the sufferer very quickly as a rule, though the more severe attacks may take several hours to subside.[5]

It is because of the danger of the allergic reactions that one should take a very careful history, ascertaining whether the recipient has ever had urticaria, asthma, hay-fever, or intestinal allergy. Some of these patients know

they are sensitive to eggs, for instance. A fasting donor, or at least one who has not eaten any of the offending foods for several hours, should be used.

I am submitting the case record, briefly, of a very striking case. Mrs. M., white American, age 23, came to the hospital Oct. 2, 1932, for a curettage because of incomplete abortion. She had been bleeding three weeks. Her temperature, pulse, and respiration were normal. She was curetted and some remnants of the placenta and membranes were removed by a colleague. Three days later, because of weakness and secondary anemia, I was asked to give her a transfusion. Her red cells numbered 2,580,000, her hemoglobin was 48 per cent, her white count showed 8,200 cells, with 72 per cent polys. Temperature normal. Her husband was found suitable and arrangements were made for the transfusion. I gave her 360 cc. by the direct method. In forty minutes after the operation she began having erythematous blotches on her face and neck. These were accompanied shortly by urticarial wheals all over her body, arms, and legs. Her lips swelled and her eye-lids almost closed her eyes. She developed a cough and complained of abdominal cramps. The itching was intense. Adrenalin chloride was administered in 15-minim doses every fifteen minutes. After the third dose, alleviation of the itching was obtained, but the wheals did not disappear for twenty-four hours. The next day her temperature ranged up to 101°, but subsided with no other ill effects.

On our rechecking this history, the patient said she always had "hives" when she ate eggs. Her husband said he had fortified himself with an egg malted milk before coming to the hospital to give the blood. I believe this reaction could have been prevented had I used a little fore-sight. This patient showed no trace of albumin or hemoglobin in her urine following the reaction, thus ruling out hemolysis as a cause of the reaction.

Karsner, in the Journal of the American Medical Association of January 6, 1921, gives the following definition and explanation of the anaphylactoid type of reaction: "It is well known that the injection of foreign protein into an animal may lead to reactions of greater or less severity, and that there is considerable individual variation within any given species. In certain instances, as for example the injection of eel serum into guinea pigs, fatal reactions may follow the introduction of extremely small amounts of the foreign protein. It is rare, however, that any individual shows reaction to a native protein from another individual of the same species, but such a possibility can not be entirely excluded. In other words it is to be considered possible that the protein of one man may be toxic for another man."

This type of reaction differs in many ways from any of the others. The cases reported all have typed perfectly, both before and after transfusion. None of them has shown evidence of hemolysis in the subsequent urine examinations. All of them have had their reactions in about one hour following the transfusion. All of them have their reactions ushered in by a severe chill followed by very high temperature. There are cyanosis, very rapid respirations, mental confusion, and in a majority of cases, convulsions. Some have required chloroform or ether to control the convulsions. The temperature reaction usually lasts for days. They never show urticarial or skin manifestations. The character, severity, and duration of reactions, the absence of incompatibility between the blood of the respective patients and donors, and the absence of any skin irritation or urticaria, usually serves to classify this type of reaction.

It is a very rare type. I have seen one case that comes under this classification. In this case the patient became sensitive after repeated transfusions from the same donor. The case history is that of a Negress aged 37 years, who came to the hospital in January, 1930, with a bleeding fibroid of the uterus. Her red count was 1,300,000, with a hemoglobin of 32 per cent. She was very weak and emaciated. Her husband was a very large Negro, and he cross-typed perfectly. I gave her 250 cc. the first time, without any reaction. This was followed in six days by another transfusion, of 360 cc. Eight days later I gave her 400 cc. more. All of these transfusions were from the same donor, and they were cross-typed before each. Her red count had mounted to 2,750,000, with the hemoglobin 52 per cent, before the third transfusion, and her general condition was very

much improved. About fifty minutes after the last transfusion the patient complained of headache. She immediately had a very severe convulsion in which her head was drawn back. Her respirations were rapid and labored. Her nails were cyanotic. This convulsion lasted about twelve minutes. When it ceased, the patient quit breathing for a full minute or more, and I could not feel her radial pulse. In the course of another fifteen minutes she was breathing fast but otherwise normally. Her pulse was full and the rate was 120. She was given adrenalin. Her temperature was 106°, and continued high for twenty-four hours. During the next thirty-six hours she had several light convulsions, and was delirious practically all the time. Morphine and adrenalin were given frequently. The temperature curve gradually came down by lysis over a period of eight days. At no time did we find more than a trace of albumin and a few hyaline casts in the urine. Her red cells were 3,650,000, with 64 per cent hemoglobin at the end of this time, and we decided to operate. Her ultimate recovery was uneventful.

I wanted to try another small transfusion from another donor, but the patient refused.

E. Traum,[6] after reviewing the literature, recommends different donors for repeated transfusions on the same patient. Apparently the skin tests for detecting sensitivity with the donor's serum have been very misleading.

In conclusion, there is indisputable evidence of the actual and potential dangers to the life of a patient from blood transfusion reactions. To eliminate some of these and to reduce the frequency and severity of others, the following precautions should be observed.

1. All typing should be done by experienced technicians, and no blood should be used simply because the donor and recipient are in the same group; they should be cross-typed.

2. Avoid methods which may produce toxic or coagulation changes in the donor's blood, as in citrate methods.

3. Carefully check the history of the recipient for evidences of allergic tendencies; use fasting donors for these, and for those who have shown allergic reactions to former transfusions.

4. The operator should perfect his technic and skill. He should familiarize himself with the danger signals of reactions and be prepared to institute prompt treatment if they appear.

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DISCUSSION

DR. VICTOR RANDOLPH (Phoenix): This is a very interesting paper on a subject in which we are all interested. Dr. Littlefield has given us the points with which we should be familiar in carrying out this procedure. I should like to ask whether he has had any case in which he noticed untoward symptoms and in which he discontinued the transfusion and continued with saline.

DR. MOTT RAWLINGS (El Paso): There are two points I should like to emphasize: the great benefit to the patient of the direct method, and the necessity of not transfusing the same patient from the same donor more than once.

DR. CHAS. S. KIBLER (Tucson): The incident of the man who took the egg drink before the transfusion brings up an interesting point. That is, whether the normal individual does not always absorb food into the blood stream. There is good evidence that any substance in contact with the body—in gastro-intestinal or respiratory tracts, in the eye, or taken by injection—very promptly goes into the blood stream. Some one has tested by injecting a live culture intracutaneously; as quickly as possible he aspirated the heart blood and cultured it. He found the cultures positive for the same organism injected intracutaneously. This proves that most injections are practically intravenous, so far as time of absorption is concerned.

DR. W. W. WAITE (El Paso): I have enjoyed this paper very much. The success of blood transfusion and the choice of technic depends on who is giving it. I have given a few off and on, usually to people without means. At the time of our last meeting here I was called out of the meeting to the hospital; I found a patient unconscious and bleeding badly. Gave her a transfusion and before we got through she was talking. That night we gave her another transfusion and the next day she was quite conscious but did not remember anything about the first one. Three or four days later we were using the donor we had used the first time; it was shortly after the noon-day meal. About four or five minutes after we started the transfusion the patient started to die; we immediately stopped and gave all kinds of restoratives, and she recovered. Later she took another transfusion with no ill ef-

fects. With regard to the whole-blood method: this is undoubtedly the best in the hands of one who is skilled in the technic and who is giving transfusions frequently. If you give only an occasional transfusion, you will get better results from the citrate method, because it is not quite so easy to make mistakes with it.

DR. F. D. VICKERS (Deming): Whenever the subject of allergy comes up, somebody always gets up and tells how sensitive people are to egg albumen. One man said he had a patient who could not wear hen feathers but could wear rooster feathers.

DR. LITTLEFIELD (closing): Wish to thank the men for the discussion. I can see no reason, if a patient is receiving blood and begins to react, why it should not be continued with saline. The saline will not do the reaction any particular good, but do not know of any bad effects from the saline. The point that Dr. Kibler made about foods going directly into the blood stream is interesting. As I recall the transfusion mentioned in the paper, it was started within forty-five minutes of the time the egg malted milk was taken. In the controversy between the whole-blood and the citrate method, I personally favor the whole-blood method for the reason stated about citrate reactions. As Dr. Waite has pointed out, it is a matter of technic. The whole thing comes down to getting the needle into the vein; after that it is just a matter of pumping.

THE PHYSIOLOGY AND PATHOLOGY OF THE THYROID

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(Read before the nineteenth annual session of The Medical and Surgical Association of the Southwest, held at El Paso, Texas, Dec. 7 to 9, 1933.)

All endocrine glands may be said to have a specific function: the production of substances essential or valuable to the organism in maintaining life. By virtue of the action of these secretions, other associated activities are from time to time attributed to the various structures in the endocrine system.

The physiology of an endocrine organ may be investigated by means of experimental extirpation, transplantation, stimulation, and administration of either the gland itself or the active principle thereof, followed by clinical and pathological observation of material wherein the organ under study evidences deviation from the normal. These procedures are especially applicable to the thyroid, wherein the axiom is illustrated that any understanding of a pathological process in a tissue must be predicated on a knowledge of the physiology of the structure involved.

Thyroid pathology is, except in cases of thyroiditis and malignancy, one not only of microscopic and gross change in structure, but also a pathology of function with resulting systemic manifestations. These functional abnormalities have their counterparts in experimentally produced physiological terms of increased or lowered glandular activity.

Complete thyroid extirpation is a difficult experimental procedure in adult animals, especially the dog, due to the presence of accessory glands and the proximity of the parathyroids[1]. Ablation in young animals is comparatively simple. It is followed by a picture comparable with that of humans suffering from glandular insufficiency. The hair and skin become dry, weight is increased, body temperature falls due to the absence of heat production in the restricted metabolism, sugar tolerance is increased and carbohydrate mobilization is slowed. If the gland is removed at an early enough age, growth and bony development are impaired. In these subjects, secondary sex characteristics fail of normal development and mentality remains infantile. Cerebral cortical tissue shows neural degeneration.[a] Spontaneous or post-thyroidectomy hypofunction in either the adult human or animal engenders a picture similar to this except that changes in growth do not occur. The skin thickens and adiposity develops. Muscle tone is lost. Tissue regeneration is retarded. There is a relative anemia.[m] Body temperature falls and the subject becomes apathetic. There is, according to Galton[2], who quotes Warfield,[w] a masked hypothyroidism without myxedema, in which the patient has weight loss, fatigue, nervousness and lack of energy associated with a lowered metabolism and helped by thyroid therapy. The classical picture of myxedema is absent.

Feeding thyroid or the administration of thyroxine, the active principle of the gland, isolated by Kendall[3], produces symptoms comparable with hyperthyroidism in man. They are increased body oxidation, tachycardia, dilatation of the peripheral vascular bed, weight loss, increased mental, nervous, and physical activity. Symptoms of hypersensitivity of the

sympathetics (pupillary dilatation, arhythmias, and gastro-intestinal disturbances) may be present.

Thyroid transplants are occasionally successful. When they live, they function as normal thyroid tissue and supply the hormone to the organism in a normal manner. This largely eliminates the theory that thyroid secretion is dependent on a neurogenic stimulus for activation.[4] The presence of epinephrine in the blood stream of the individual in amounts sufficient to cause any systemic effect is highly questionable.[5] To allocate the place of a thyroid stimulant to the adrenal medulla on the basis of the epinephrine secreted into the circulation[6] is at the present time somewhat fantastic. It seems wiser to consider the stimulus for thyroid secretion to be either a real or potential hypothyroidism, as does Plummer.[7] It is possible that fright, anger, and emotion may stimulate thyroid excess function, as Crile[8] and others[43] claim, but these may cause primary thyroid stimulation rather than an indirect excitation through adrenal influence.

The known function of the thyroid, to regulate body catabolism and anabolism and keep them in proper balance, may be due to an electro-chemical mechanism, as suggested by Crile[9], or may be a catalytic phenomenon, as advocated by Mayo and Plummer.[7] By virtue of the ability of thyroxine to influence the oxidative processes of the body, the thyroid plays an important part in the regulation of body temperature[10] the mobilization of carbohydrates, nitrogen balance, and calcium excretion.[39] According to Kendall the function of the thyroid is to "regulate the capacity of a cell to use oxygen[40]."

Much has been written concerning the influence of the thyroid on the endocrine system. It remains, however, clouded in the realms of speculation. Statements of facts cannot be made in this matter until research has untied many an endocrine knot. It is unwise to hazard opinions on glandular interrelations when so little is known concerning such dramatic structures as the adrenal medulla, the thymus and the testes.

Crile[11] has expressed opinions concerning adrenal-thyroid correlations. He believes there is a reciprocal action between the thyroid, the adrenals, and the nervous system, on a physiochemical basis in which the thyroid is activated by the sympathetics. He draws the conclusion that the adrenals (being the brain of the sympathetic) are most important in determining thyroid activity. He also states that the thyroid, through control of the permeability of the body cells, governs the rate of cellular activity so that, in increased thyroid function, cell action is accelerated.

The thyroid's influence on liver function has received considerable study. Simonds and Brandes[12] found the livers of dogs fed thyroid substances to be almost glycogen free. There is apparently no serious impairment of liver function accompanying this change[16], although John[13] quotes authority that the hyperthyroid liver shows atrophy annd extensive fatty degeneration. Asher found the hyperthyroid liver cells able to form glycogen but unable to hold it.

Whether changes in sugar metabolism are to some extent manifestations of endocrine reaction to thyroid secretion, is debatable. It is known that removal of the thyroid lowers blood sugar, that feeding thyroid produces a hyperglycemia and that thyroid extract acts synergistically with adrenalin in increasing adrenalin hyperglycemia. Over half in one series of hyperthyroid cases, showed impaired sugar tolerance. This may be due to increased mobilization of glycogen, hyperadrenalism as a result of adrenal stimulation, or the effect of throwing a heavy load on a partially exhausted prediabetic insulogenic system.[13]

The parathyroids and thymus, due perhaps to their proximity, have often been allegedly changed by thyroid pathology. It is known that thyroid extract is of value in tetany[14] and that thyroid-fed animals excrete large amounts of calcium[15]. Some authorities state that thymic hypertrophy occurs in thyroid feeding and early involution follows thyroidectomy. Not enough work has been done to settle the matter of thyropituitary relationships. The literature states that the anterior lobe of the pituitary will hypertrophy if the thyroid is removed.

That gonadal function to some extent depends on thyroid action seems clear. Myxedema frequently follows the menopause; ovarian hypertrophy after thyroidectomy is common. Thyroid enlargement at puberty and gonadal failure in cretinism are well-known entities.

As Menne[17] states, neither clinical nor experimental data are sufficient to date to settle the question of the direct effect of the active principle of the thyroid gland on the heart muscle in hyperthyroidism. Andrus[18] has shown in an animal which has been fed on large amounts of thyroid extract that, after isolation of the heart, the tachycardia remains, the oxygen-combining power and lactic-acid content of the muscle is increased, the glycogen content lessened. The loss of glycogen from cellular elements renders them more liable to injury[19], while increased oxygen and lactic-acid content are indications that the myocardium is participating in the increased metabolism of the body. Yater[23] has noted the fact that in a perfused heart thyroxine increases its metabolism. That the tachycardia is a reaction of cardiac muscle and not the result of sympathetic stimulation has been shown by crucial perfusion experiments[20 21 22].

The weight of recent opinion tends to discountenance the effect of thyroid secretion as a direct factor in organic heart disease[36]. Weller et al[24] and Rake and McEachern[19] found no gross or pathological changes in exophthalmic goiter hearts which were not equally represented in a control group.

It is rational to believe that a heart, laboring under the demands of a circulation attempting to meet the needs of a body with a greatly increased metabolism, may exhaust its nutrition and show evidences of insufficiency[35], particularly when its own myocardium is participating in the increased metabolic processes[37].

Goiter classifications rival those of nephritis in multiplicity. The majority of these are differentiated by the presence or absence of toxicity or nodules. An example of the former is cited by Moulton[26], who makes the elemental classification of goiters associated with normal, hypothyroidism, or hyperthyroidism. Marine's classification is one based on the pathological picture: hypertrophy, hyperplasia, colloid, exhaustion atrophy, fetal adenoma and malignant adenoma. Van Meter[27] quotes a classification in which is a combined functional and pathological division, as nontoxic or toxic diffuse, nontoxic or toxic nodular.

Hertzler's[25] simplified grouping seems broad and practical. To him the division of the disease into groups illustrates the progress or stage of a single pathological process. He presents pathological and clinical material to justify this stand. His classification is (1) colloid; (2) adenoma without toxic symptoms; (3) adenoma with toxic symptoms; (4) Graves' disease (exophthalmic gland).

Hertzler states that he has, through many years' study of individual cases, seen one patient run the gamut of simple colloid, nontoxic adenoma, toxic adenoma, Basdow's triad, and exitus through heart failure. The stage in diagnosis is referred to as one would in syphilis refer to a papillary syphilid or gumma. If this conception can be substantiated by clinical data, it renders thyroid pathology clearer and makes the goiter of adolescence a less innocent thing than it is considered to be.

Histologically the normal thyroid gland consists of acini roughly polyhedral in shape, averaging 0.3 to 1 mm. in diameter, lined with cuboidal epithelium and filled with a gelatinous colloid. Between the acini are groups of cells resembling the epithelium lining the acinal vesicles.. Small connective-tissue trabeculae form a skeleton upon which these cells rest. Grossly the gland is smooth and regular in outline.

The commonest change in the thyroid is one of symmetrical enlargement. The lobe outlines remain uniform, but histologically the acinar compartments are increased in size and filled with additional colloid. The cuboidal cells of the lining are elongated and flattened. There is no increase in glandular proliferation, but the interstitial cells may have disappeared.

Nodular thyroids, or the so-called "adenomas," may be noduular grossly or discovered as such only on histological examination. The gross nodular, or to use Hertzler's term "bosselated," enlargements are knotty or lumpy thyroids in which new acini develop in colloid goiters as a budding process from the epithelium of old acini or by proliferation of interacinal cell groups. The colloid seems unchanged. The newly formed acini are lined by flattened cuboidal epithelium.

Multiple new areas are found in these adenomatous or bosselated glands. In fetal adenomas the growths are isolated and surrounded by a capsule, which separates them from normal tissue. Histologically they resemble the

fetal gland, containing but little colloid and many small acini. They seldom form papillary outcroppings, are of limitless growth, and easily degenerate or become malignant. They rarely become toxic.

The irregular new growths may be devoid of any functional activity or may be compensating for decreased function in the rest of the gland. At this time they are termed non-toxic. When they become active or their activity exceeds the normal requirements, toxicity may be attributed to them. Toxic nodulations on microscopic section frequently show increased vascularity, the cuboidal cells become spherical or hypertrophic, the colloid is granular, with cellular debris scattered through it. Papillary growths may appear sprouting from the epithelium into the colloid and many new acini are seen forming in the interstitial spaces.

The epithelium of the exophthalmic gland is usually columnar. Papillary outcroppings from the acinar linings may be so marked as to almost obliterate the colloid[28]. Epithelial proliferation is extreme and new acini crowd the interacinal spaces. Grossly the gland is granular and firm without nodulations. It is deep red on cut surface.

Graham's classification of malignancies of the thyroid gland, as quoted by Dinsmore[29], lists: first, lympho and spindle-cell sarcoma, the latter the most malignant of all thyroid tumors; second, mixed carcinosarcoma; third, scirrhus carcinoma, adenocarcinoma (the former 100 per cent fatal, the latter least malignant—neither found in the adenomata), papillary carcinoma and malignant adenoma (both discovered in adenomata). The papillary carcinoma metastasizes very slowly, whereas malignant adenoma is the most frequent cancer of the thyroid, with early metastasis through the blood stream.

Hertzler would include fetal adenomas as true neoplasms of the thyroid, inasmuch as they are isolated nodules, unicentric in origin, tend to limitless growth, and with age degenerate into cysts, become mildly toxic, or malignant. He states that, in 300 malignant goiters, nine of ten were from fetal adenomas.

The place of the adenoma in goiter pathology is a subject of controversy. Wolfer's theory is that they are developed from embryonic rests. Graham[4] feels that all nodular goiters might be classified with neoplasms similar to adenoma of the breast and fibromyoma of the uterus. He thinks they are due to a process of irregular hyperplasia and involution. Rienhoff[30], in a meticulous study of wax reconstructions of human thyroids, came to the conclusion that Wolfer's interpretation of commonly called fetal tissue was a technical error and that there are no fetal or epithelial rests in normal or exophthalmic glands and that the persistence of fetal tissue does not occur.

Inflammatory conditions of the thyroid may be either acute or chronic. Acute thyroiditis may accompany any infectious process elsewhere and resembles inflammation in any other tissue[31]. It usually does not go on to suppuration. In event of abscess formation, hypothyroidism from gland destruction may appear.

Riedel's disease[32] and struma lymphomatosa of Hashimoto are manifestations of chronic thyroiditis. In both there is marked lymphocytic infiltration of the glandular stroma with connective tissue deposits which go on to degenerative sclerosis of the entire gland. In struma lymphomatosa the gland grossly is white, firm and resembles a fibromyoma[33]; in Reidel's disease it is hard, friable, and looks like beefsteak.

In both conditions the thyroid is hard, but whereas in struma lymphomatosa the glandular outline is not lost, in Riedel's there is a woody induration, not only of the thyroid, but also of the contiguous cervical tissues, resulting in loss of lobe outline.

The etiology of chronic thyroiditis is unknown. Graham feels that there is no relation between the two conditions but that there may be some association between struma lymphomatosa and lymphosarcoma of the thyroid[34].

To summarize then: The thyroid is a gland capable of unusual functional and pathological abnormalities. In its normal state it is, perhaps more than any other single tissue, the balance wheel of the body. By virtue of its physiology, and its correlation with the other endocrines, it serves not only to determine structure and physique but also so to affect mentality, fatigue, and energy as to determine the personality of the individual.

* * * *

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Doctor H. F. Helmholz, Chief of the Division of Pediatrics at Mayo Clinic and Professor of Pediatrics at the University of Minnesota, was a luncheon guest of the El Paso County Medical Society, January 20th. Dr. Helmholz spoke on "Urinary Infections in Childhood."

WHAT EVERY WOMAN DOESN'T KNOW—HOW TO GIVE COD LIVER OIL

Some authorities recommend that cod liver oil be given in the morning and at bed time so as to assure an appetite for the oil, while others prefer to give it after meals in order not to retard gastric secretions. If the mother will place the very young baby on her lap and hold the child's mouth open by pressing the cheeks together between her thumb and fingers while she administers the oil, all of it will be taken. The infant soon becomes accustomed to taking the oil without having its mouth held open. Mead's Newfoundland Cod Liver Oil, of minimum acidity and prepared from fresh healthy livers, is well tolerated by infants and children and is palatable without flavoring.

If given cold, cod liver oil has little taste, for the cold tends to paralyze momentarily the gustatory nerves. As any "taste" is largely a metallic one from the silver or silver-plated spoon (particularly if the plating is worn), a glass spoon has an advantage.

Mead's 10 D Cod Liver Oil is made from Mead's Newfoundland Cod Liver Oil. In cases of fat intolerance the former has an advantage since it can be given in one-third and one-half the usual cod liver oil dosage.

SURGERY IN TUBERCULOSIS OF THE THYROID

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(Read before the nineteenth annual session of The Medical and Surgical Association of the Southwest, held at El Paso, Texas, Dec. 7 to 9, 1933.)

Tuberculous goiters have included some of the most desperate operative risks that have come into my thirty years' experience with thyroid surgery. One of these patients came to me on a stretcher and the degree of toxemia was so extreme in five that the many-stage operation seemed the only safe procedure. The basal metabolic rate ranged from 40 to 68; exophthalmos was pronounced; the apex beat of the heart ranged as high as 172 on the operating table and tremor was extreme. Ligations were done in two cases in my earlier experience, followed in one case by rapid partial excision and pack, with secondary closure. Only one patient was within normal limits as to pulse rate and basal metabolic rate, while two were moderately toxic. The degree of hyperthyroidism apparently has been considerably higher among my patients and the percentage of highly toxic cases greater—as also with many other American surgeons—than in series reported from foreign countries.

Fortunately thyroid tuberculosis is of relatively infrequent occurrence though it is of much interest and, because of the valuable lives concerned, it is of great importance.

This series includes eight cases from over 6,000 goiters under observation during thirty years. A series of seven cases was reported by Plummer and Broders[1], increased to twenty-one by Rankin and Graham[2]. These reports indicate the small number of tuberculous goiters.

CLASSIFICATION

Many classifications are given by different writers. Probably as simple and good as any is that of Wegelin[3], whose experience with thyroid pathology as professor at the University of Berne, Switzerland, is very great. He classifies acute and chronic tuberculosis of the thyroid, and states that most frequently the infection is hematogenous. Occasionally it is apparently primary as shown by postmortem examination, but probably actually metastatic in origin. Three forms of chronic tuberculosis are mentioned: (1) proliferating form, which is most frequent; (2) caseating nodules with abscess; and (3) sclerosing tuberculosis, sometimes mistakenly diagnosed as Riedel's iron-hard struma. Tubercle bacilli are seldom demonstrated: the pathological diagnosis is based on finding characteristic microscopic tubercles. The acute form is well illustrated by a case reported by Moschowitz[4], in which there was dysphagia, loss of weight, and fever up to 104.5°. This patient had a hard mass which was diagnosed as diffuse miliary tuberculosis.

Illustrating the sclerosing form, Budd and Williams[5] report three cases with a gross appearance of malignancy. There were, however, the symptoms of hyperthyroidism with loss of weight, tachycardia, high basal metabolic rate and nervousness. Probably Neuhof's case is of the same class: he reports[6] a young adult with dypnea, dysphagia, basal metabolic rate normal, no symptoms of hyperthyroidism, diagnosed as malignant. The growth was rubbery in consistency; the microscopic diagnosis was miliary tuberculosis. Meyer's[7] cases illustrate both the sclerosing form and those with abscess. He states that the symptoms resemble chronic thyroiditis or malignancy, or both; that cold abscess is less frequent; that rapid pulse rate, loss of weight, and other symptoms of exophthalmic goiter are not so frequent. My own cases include two with a pathological diagnosis of exophthalmic goiter and tuberculosis; three, adenoma associated with tuberculosis; and three of the sclerosing form, these cases giving the symptoms of exophthalmic goiter though not the microscopic picture.

It is interesting to note that two great authorities of earlier years believed that tuberculosis and goiter were never associated. Rokitansky[8] stated that the thyroid is never affected by tuberculosis. Jones[9] quotes Virchow: "The presence of goiter is proof that the subject was not tuberculous." He also states that in general miliary tuberculosis there are few thyroids involved. Extensive tuberculous involvement of the lungs or other important organs does not seem commonly to accompany thyroid tuberculosis. In speaking of

blood-stream or other metastatic origin of thyroid tuberculosis, Wegelin states that it is usually impossible to locate the original focus of infection.

DIAGNOSIS

A diagnosis of tuberculosis of the thyroid is seldom made without a microscopic examination. Exophthalmic goiter is the diagnosis most frequently made after examination and obtaining a history of the symptoms of the patient; slightly less frequently, toxic adenoma; cancer, simple thyroiditis, and Riedel's struma occasionally. The diagnosis on gross pathological examination of the tissue removed is also almost always incorrect, but has coincided with the preoperative diagnosis in all of my own cases but one, in which a diagnosis of thyroiditis and adenoma was made postoperatively in a case preoperatively diagnosed as adenoma only. Practically all writers agree as to the impossibility of preoperative diagnosis. The important point regarding diagnosis has been that surgery was definitely indicated in all cases. It seems highly improbable that any of these patients would have recovered without operation, certainly not with as full a measure of health as they have obtained following operation.

RESULTS

All of my patients have made satisfactory recoveries from their operations in spite of the fact that the situation seemed desperate in over one-half of the cases. Partial bilateral excision, removing only evidently diseased tissue, is usually indicated, but stage operation may be necessary, as before mentioned. Electro-surgery seems specially important in order to seal the rich blood and lymphatic supply and prevent metastasis. One patient died from apoplexy five years following the time when she first came under my care. The remaining seven were living and reasonably well at the last report. All but one patient are from five to twenty years past their operation. The gain in weight has been striking in most of the cases, amounting to 66 pounds in one patient who was much emaciatetd when she was first seen.

It is evident that, in the interest of permanent cure, general health measures are approximately as important as surgery. Most careful attention should be given to all those hygienic, dietetic, and climatic influences which have proved so vaulable in the cure of pulmonary and other forms of tuberculosis.

SUMMARY

Tuberculous thyroiditis may be either an acute or, more frequently, a chronic inflammatory process, which may go on to fibrosis or suppuration.

In the earlier subacute stage the symptoms may be indistinguishable from those of extremely toxic exophthalmic or adenomatous goiter; later on malignancy may be suspected.

A diagnosis is seldom possible without microscopic examination.

Surgical treatment gives excellent results in suitable cases though careful preoperative and postoperative care and frequently the stage operation, are essential to success; electro-surgery elimniates the danger of metastatic spread by sealing blood and lymphatic vessels.

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THE COMMON COLD

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(Abstracted from an address before the Las Vegas Kiwanis Club at its Annual Ladies' Night, December 6, 1933.)

Until recently, the common cold has received very little attention and consideration from scientific medicine. This neglect has probably been due to the fact that most individuals recover from their colds without complications, in spite of treatment that often is contrary or even injurious, and that 35 per cent of all colds recover with no treatment whatsoever.

Lately, the common cold is being recognized more and more as a predisposing cause of various more serious infections and as exacting a terrific toll in suffering and money. Amassed statistics show that the germs which enter and leave the body through the mouth

and nose cause 92 per cent of all the deaths from communicable diseases, and it may well be said that the Achilles' heel of the modern man is his mouth. Of these communicable diseases the common cold, or coryza, is the most frequent. It is responsible for a greater loss of time from school and work than any other single cause. The average person is partially disabled from two to six weeks each year due to colds, and it has been estimated that colds cost the people of the United States one billion dollars a year.

Too often do we hear, "Oh, it's only a cold." That may or may not be the correct diagnosis, as 50 per cent of all disabling diseases—such as influenza, measles, whooping cough, scarlet fever, and even tuberculosis, kidney disorders, and meningitis—frequently start with a sore throat or cold, and we must not overlook the seriousness of complications. Colds frequently are followed by serious illnesses; if the patient's resistance is low, any part of the respiratory tract may become involved, resulting in serious diseases of the nose, throat, and lungs.

If, through ignorance or carelessness, the individual blows his nose improperly, he may force infected material into the sinuses and start sinus disease, or through the eustachian tube to cause an inflammation in the middle ear. Many deaf people can trace their deafness to a cold; and acute inflammation of the eyes is frequently brought about this way.

It is evident that a whole train of troubles lie in the wake of a neglected cold. Even if it is just a cold, it is not to be taken lightly. What is more, it is very easy to give a cold to someone else and the cold the other person catches from you may result disastrously. Many a person with a weak heart has died from the effects of a common cold.

The seriousness of a neglected cold is testified to by the following epitaph found on an old tombstone in a New England cemetery:

"Here lies my wife, Samantha Proctor,
She ketched a cold and would not doctor.
She could not stay, she had to go.
Praise God from whom all blessings flow."

Before considering the discussion, it might be well to consider the question, "What is a cold?"

Cecil, in his excellent "Textbook on Medicine," states that "the common cold, or coryza, is a catarrhal inflammation of the upper respiratory tract, which results from infection by micro-organisms (germs) and perhaps from other causes not yet definitely determined." This sentence just about sums up all that we know definitely about the common cold.

Our grandmothers knew in their time that a cold ran a definite cycle, for they used to say, "two days to come, two days to stay, and two days to go."

This is frequently an understatement, but we know that a cold has three distinct stages. In the first stage, the nasal mucous membrane is congested. There is sneezing inability to breathe through the nose; the head feels heavy and stopped up; the eyes burn; the skin is dry; there is chilliness, possible fever, aching of the back and legs, and general discomfort. This stage lasts from one to three days.

The second stage is characterized by marked secretion and discharge of watery material. Areas of excoriation appear around the nose and mouth. The sense of smell, taste, and hearing are often impaired. Constipation is common. This stage lasts from two to seven days.

In the third stage, the watery discharge changes to a thick mucous, or muco-purulent, discharge and gradually subsides.

The results of the intensive research work done upon the common cold during the past eight years show that a cold is the same regardless of climate; that most people have two or three a year and that wage-earners of this country lose from one to three days a year from colds; that sex has nothing to do with the incidence of colds, but there is a class definitely susceptible to colds; that over two-thirds of the people have abnormalities or diseases of the nose and throat; also that occupation has a bearing on susceptibility, e. g., office-workers have nine times the colds that taxi-drivers have and four times as many as United States Army men.

Weather conditions and temperature changes markedly affect the prevalence of colds; a drop of ten degrees in the weekly average temperature was accompanied by an increase of eighteen colds per week among a group of 6,700 under observation. The name of the

disease "cold" implies a connection between weather and the incidence of colds.

Colds have a seasonal distribution and occur most frequently in the dark cold periods of the year. The most striking feature of the seasonal distribution is the high prevalence in the autumn, followed in the winter and spring by a series of epidemics. The incidence is not, therefore, as might readily be supposed, equally spread over the winter months, but consists of a series of distinct epidemics irregular in spacing and magnitude.

Age affects the incidence and severity of colds. The incidence of respiratory disease is at its highest in children under five years, declines progressively to about the age of fifteen to nineteen years, increases somewhat between twenty and twenty-five, and thereafter declines slowly as age increases.

The average person is partially disabled forty-nine days per year.

CAUSE

To the question as to what causes a cold, you may get any kind of answer you want, from the thoughtful hypothesis of an eminent scientist to the whim of a crank with an "idea." Dr. Volney S. Cheney, Medical Director of Armour and Company and Chairman of the Valmora Sanatorium Medical Committee, is convinced that colds result from an acid condition of the body which predisposes to infection with a filtrable virus (a germ so small that it escapes the finest filter known and has never been seen).

Other eminent doctors attribute the cause of colds to unclean city streets, to carrying of the germ by cats and dogs, to purely bacteriological causes; and one even blames bedbugs, while another claims that women take cold from powdering their noses too much. There is still another theory, that colds start with a disturbance of the heat-regulating mechanism of the human body.

The predisposition to catching cold can be either congenital or acquired. The acquired may be permanent or temporary. If a predisposed person is chilled for a considerable period of time, there occurs, first, a contraction of the blood vessels of the skin and of the respiratory-tract mucous membranes. This is normal, but if the blood vessels do not relax within twenty minutes and allow the blood to flow back into these membranes, it is abnormal. This long period of greatly reduced blood circulation reduces the defense against bacteria always present on the mucous membrane, and when these bacteria or the filtrable virus gain the upper hand, inflammation occurs, i. e., a cold results.

Colds may also result from the congesting action of dry hot air, of certain gases, or of dust; and there is much evidence that susceptibility to acquiring colds is influenced by the state of the nervous system and of the mind.

The heat-regulating apparatus of normal persons may be put out of action temporarily by a fall into ice-cold water, by alcohol or narcotics, and under such conditions they may "catch cold." A draft of air that is not felt is particularly dangerous because it does not excite the heat-regulating apparatus to action.

While there is no doubt that the predisposed person can so train his blood vessels, by being much in the open air, that they learn to react to cold in a normal manner, it would be very much better for this hardening process to be carried on under the direction of the family physician.

Extensive research work has been conducted for the past eight years. But the common cold remains almost as mysterious a disease as sleeping sickness and cancer, and, as yet, its cure and cause have not been found. Overeating, indigestion, constipation, poor ventilation, irritation of the mucous membrane of the nose and throat due to smoky or dusty air, fatigue, lack of out-door exercise, and loss of sleep, are often referred to as the main causes of colds.

While the definite cause of a common cold is not known, we do know that a cold is infectious and is now widely believed to be caused by a filterable virus or invisible germ. This invisible germ which causes you to catch cold, may enter your nose and throat from the air when an infected person talks, coughs, or sneezes. You may catch cold by kissing or by using an unwashed glass, spoon, or fork, which has been used by someone with a cold. The germs may be on your hands because you have shaken hands with someone with a cold or have touched something which has been handled by a person with a cold. A soiled

handkerchief of the individual with a cold is a constant menace, particularly if the secretions have dried and can float around in the air.

The great Dr. Johnson poked fun at a Scottish historian who noted that the inhabitants of the Scottish island of St. Kilda went through the winter without colds and developed them only in the spring (when mainlanders came over). We now know that small colonies of people, when completely isolated from the outside world, are completely free from colds as long as they remain isolated, no matter to what extremes of exposure and cold they are subjected.

In a recent study of the common cold in an isolated arctic community, it was noted that there was an almost total absence of disease of the upper respiratory tract until forty-eight hours after the arrival of the first boat in the spring. Then, within a week, 25 per cent of the inhabitants were stricken. No significant change in the type of bacteria of the upper respiratory tract could be demonstrated at this time.

Today, almost everyone believes that colds are infectious, i. e., caused by germs. You can sit in wet clothes by an open window in January and, if there are no cold germs present, you won't catch cold. The chances are, however, that cold germs are present, and in your own nose and throat. Most people in large communities carry a quota of cold germs with them all the time. When their resistance is lowered by wet feet or by exposure, they succumb to the germ and "catch cold." It is the germ which causes the cold, however, not the wet feet.

PREVENTION

1. Avoid people with colds. During flu epidemics it is wiser to stay at home than unnecessarily be part of a crowd in poorly ventilated rooms, street cars, or meeting places.

2. Sleep eight hours at least, with windows open in winter and summer. Cold air is not necessarily fresh air.

3. Get plenty of fresh air, but avoid drafts. Live and work in well-ventilated rooms. It is better for the thermometer to read 68 degrees than over 70. Overheated rooms cause more colds than underheated. Windows should be opened at least twice a day, even during the winter, for complete change of air. Colds are due to foul, stuffy air, with the germs which such air is almost certain to contain, rather than to cold pure air. If there are dust or fumes where you work, wear a mask, if possible—at least clean out your lungs with fresh air for a few moments at your lunch hour or when walking part of the way to and from work.

4. Practise deep-breathing exercises.

5. Eat nourishing food. Avoid overloading of the stomach (particularly by fat meats, gravies, fried foods, richly spiced foods and rich or sweet desserts). The addition of high concentrations of vitamins A and D to the diet does some good.

6. Keep your bowel movements regular.

7. Drink plenty of liquids, including milk and fruit juices. Drink at least six glasses of water a day.

8. Take plenty of exercise, indoors and out-of-doors in the sunshine, every day.

9. Avoid undue fatigue.

10. Brush your teeth at least twice a day. Wash your hands before eating and do not put fingers in nose or mouth.

11. Keep the nasal passages clean and moist. If necessary, use some sort of mild spray or douche.

12. Bathing each morning with water cool enough to give a healthy glow after the rubdown, is a good practice. Cold baths, massage, and vigorous rubbing, will harden the skin to resist variations in temperature. If cold water is too much of a shock at first, the temperature can be lowered a little each day.

13. Wear sensible clothing. Wear enough clothes to be comfortable. In this day of overheated houses, offices, and shops, it is better to put on extra wraps when going out of doors than to wear heavy underwear all of the time. Winter shoes should have thick soles, and rubbers should be worn on rainy days. Wear wool stockings in winter.

14. Breathe through your nose. Air is filtered as it passes through the nose on its way to the lungs. The mouth breather takes dust and germs directly into his throat. If you have trouble breathing through your nose, see your doctor.

15. Have diseased tonsils, adenoids, or bad teeth removed. Make sure there are no obstructions or infections in the nasal passages.

16. Keep your feet dry. An extra pair of shoes and stockings kept at your place of work will add to your health and comfort when you are caught without rubbers. Rubbing the feet briskly with a rough towel will help you keep from taking cold from wet feet.

17. Cool off gradually. If you are perspiring, an electric fan or a cool breeze may start a cold.

18. If you keep on taking cold in spite of taking care of yourself, see a doctor. If you take cold often, if colds hang on, if you have a persistent cough, your health needs watching. The proper treatment of colds will prevent much sickness and many deaths from influenza, pneumonia, and other serious diseases.

The consensus of medical opinion is that at present no one has any right to proclaim on scientific grounds that he has any means of warding off a cold. But man, and especially man with a cold, lives in hope. At present there are four more-or-less scientific means of preventing colds: vaccination, ultra-violet rays, chlorine, diet. Each of them has its supporters, its detractors, its triumphs and its failures.

Cold vaccines are of two types: autogenic and stock. Both are made up from the eight bacteria which seem to play a secondary part in causing colds. In the autogenic type, the bacteria are taken from the nose and throat of the individual who is to be vaccinated. Though such vaccines are theoretically superior, they are considerably more expensive. Stock vaccines, whose bacteria are garnered from any handy nose and throat and laid away till wanted, are prepared by the drug manufacturer. There are several high hurdles which must be taken before vaccination-for-colds can approach success. The consensus of medical opinion today is that vaccines have little value.

The use of artificial sunbaths and ultra-violet rays, in preventing colds, has aroused considerable skepticism in the medical profession. Hereto, the general opinion is that there is no special merit in them. However, Drs. Smiley and Maughan, at Cornell University, make the sun lamp a cornerstone of their extremely effective cold-prevention class. They think it tones up the skin capillaries so that the patient becomes hardened to drafts, but they do not depend upon sunbathing alone, nor do they claim more for it than that it keeps the patients interested in the cold class.

While the influenza epidemic of 1918 was raging, cold students noted one minor fact with major interest: workers in plants where chlorine gas was much in the air were practically immune to the disease. The war had already taught a few alert army doctors that chlorine gas not only kills men but also gives them relief from colds. Ever since, there has been a small but earnest school of chlorine inhalationists. Chlorine inhalation became a fad and swept over the country five or six years ago, but the "chlorine parlors" run by amateurs were long ago suppressed as much too dangerous.

Scientific medicine regards chlorine with the special skepticism which it reserves for treatments which have once been fads. There is only one experiment of any scientific standing and it will require further investigation to determine the value of chlorine used in the treatment of colds.

The researches of Valmora's Dr. Volney S. Cheney and of Cornell's Drs. Smiley and Maughan, led them to believe that colds result from an acid condition of the body. To overcome this, they prescribe various alkalies—among them, bicarbonate of soda—and diet.

There are certain general rules for eating which are unanimously accepted by cold students. To ward off colds, everyone agrees, you should get plenty of vitamin A, which is found in orange juice, tomato juice, codliver oil, haliver oil, viosterol, prunes, spinach, carrots, and other depressing foodstuffs. Also get plenty of vitamin B, which plays the part of alkaline pills in combating acidosis. Vitamin B occurs in whole cereals, fruits, nuts, and vegetables. Very little candy and sweetstuffs and very much water (at least six full glasses daily) are also advised.

This sort of dieting is a cornerstone of the New York Federal Reserve Bank's extremely effective anti-cold measures. By making use of diet, sunbaths, and inoculation, and sending home snufflers with any trace of fever, the bank has come closer to whipping the cold problem than any other large downtown office. A recent grip epidemic showed a seven per cent peak, as against a peak of from fif-

teen to twenty-five per cent in the rest of the Wall Street District.

The Federal Reserve diet, like the diet urged on Cornell students by Dr. Smiley, is alkaline, non-fatty, non-carbohydrate, strong in fruits, vegetables, and fluids.

Whatever the cause of colds, colds are a disease and human beings are more susceptible to disease when "run down," below par. Hence it is obviously sound practice to keep in good condition to avoid taking cold. At present, the most effective preventive is the isolation of persons suffering from a cold. Dr. Britton, chief surgeon of the International Harvester Company, who is also a member of the Valmora staff and is one of Chicago's leading chest specialists, states that the greatest single thing to be done to stop colds would be to send home anyone showing up at work or school with a running nose. No one ever got a cold by himself. One catches it from some cold carrier by being breathed upon, sneezed upon, or coughed upon. Until the innocent bystanders rise up and bodily eject the snifflers and sneezers from school, office, theatre, and restaurant, until bed becomes the only place where a cold carrier can peaceably exist, the American people will continue to catch some 100,000,000 colds every winter.

TREATMENT

One of the doctors engaged in research upon the common cold, when asked what he did for his colds, replied gruffly, "I go to bed and use two dozen soft linen handkerchiefs." This would indicate that our knowledge of the treatment of a cold is limited. We all know that there is no specific for the treatment of colds and until the specific causative factor is discovered, the treatment will remain essentially symptomatic.

One of the main symptoms of a cold is an acid condition of the body. To counteract this acidity, fruit juices and alkalies are used. Another prominent symptom is congestion of the lining membranes of the nose, throat, and bronchi. To relieve this internal congestion, free catharsis is produced and thus the blood vessels of the congested area are emptied by lowering the blood pressure, the fever is reduced by various means, and the blood is brought to the surface of the body and perspiration produced. These procedures help when one has a cold, but let me repeat again that no doctor can cure a cold at present. All that we can do today is to prevent infection, sometimes to abort a cold, to relieve suffering, and to shorten the length of time that a cold lasts.

OUTLINE

A. Measures that have Value in all Stages of a Cold.

1. Rest in bed, for a day or so. There is no question but that staying in bed is the closest we have come to finding a panacea. Everyone, from your grandmother to the president of the American Medical Association, agrees that bed is the place for one with a cold.

2. Take some mild laxative—the salines are best: epsom salts one-half ounce, or one to four drams, magnesium citrate, twelve ounces (one bottle).

3. Produce free perspiration by taking a hot general bath, a hot foot bath, or a Dover's powder. As soon as you have rubbed yourself dry, cover up in bed with two extra blankets so that you can sweat the cold out of your system. To help you sweat, take a hot drink such as hot lemonade or a hot "toddy" of whiskey, sugar, and hot water. Brandy or whiskey with a small cupful of hot black coffee or tea is also very good. These not only help you to sweat, but relieve the general body weariness and the feeling of apprehension common to a cold. A word of caution: overindulgence in alcohol will lower the resistance and pave the way for pneumonia.

4. During a cold the blood tends to be less alkaline than usual. To combat this acidity, take one teasoonful of sodium bicarbonate, (baking soda) in water or lemonade every four hours. In addition, take phenacetin, or aspirin, grs. V., with the soda to relieve discomfort. Repeat every three or four hours if necessary.

5. Eat lightly of simple, nourishing food. Nothing could be further from good advice than "feed a cold and starve a fever." A cold is a fever, the inflammation being localized in the respiratory mucous membranes. Overloading the stomach is harmful, and meats, gravies, fried stuff, and richly spiced foods are especially to be avoided. Plenty of cold water, milk, and fruit juices are advisable. Take at least one-half glassful every hour while awake. According to recent research, the addition of

vitamins A and D apparently are not of much help, but are worth trying.

6. Gargle with warm salt water and soda three to four times daily. A teaspoonful of ordinary table salt and one-half teaspoonful of baking soda to a pint of water is an ideal mixture. This can also be used as a nasal douche.

7. Avoid any exposure to cold. Cold baths, cold showers, and strenuous exercise are valuable hardening methods in the intervals between colds, but should be stopped while one has a cold.

8. Smoke and cold damp air should not enter the respiratory tract.

9. Isolation is desirable, as it protects others and lessens chances of catching something else while your resistance is low.

10. Blow your nose properly and as little as possible, or you may get mastoid or sinus infection, for 10 per cent of all colds develop sinus trouble. Hold your handkerchief over the bony bridge of the nose so as not to constrict the nostrils. If the nostrils are compressed, the air pressure is increased and the discharge may be blown into the sinuses, middle ear, or the eyes. When the laborer puts his thumb over one nostril and blows out the other, he is very apt to spread the discharge from the compressed side to the sinuses, ear, and eye.

B. The Beginning Cold. In addition to the ten measures listed under A, try to check the infection by local application in the nose and throat of 20 per cent argyrol, or a 1:5000 solution of metaphen merthiolate, hexylesorcinol, or any other mild antiseptic. A few drops are introduced into each nostril with a medicine dropper and allowed to trickle back through the nose until the whole naso-pharyngeal mucous membrane is well covered. Do not blow the nose, but absorb any excess discharge with gauze, so as to keep the solution in contact with the membranes as long as possible. This procedure may be repeated several times a day.

C. The Second Stage of a Cold. In the second stage, when there is a profuse watery discharge, the inhalation of some soothing preparation is often very beneficial. A mixture of thymol, menthol, eucalyptol, and mineral oil, inhaled from an atomizer, is soothing and healing. The steam inhalation of tincture of benzoin and the oil of pine needles, helps the same way. (With the latter mixture the housewife should be forewarned that it is difficult to clean the utensils used.) A teaspoonful of the above mixture is put in a teacup and filled with boiling water. The face is placed low over the cup at once, with the entire head and cup covered with a towel. Inhale deeply for a few moments through both nose and mouth, and be careful to avoid exposure for at least one hour after this treatment.

D. The Third or Last Stage of a Cold. In the last stage of a cold, when the discharge has become thick, alkaline antiseptic solutions used locally as a douche are beneficial. A teaspoonful of boracic acid added to the salt and soda solution usually proves sufficient. It should be snuffed into the nostrils from the hand, from an atomizer, or from a nasal douche.

E. Other General Measures That May Be Tried. The vaccine treatment for recurrent colds helps only in prevention or prophylaxis, and works only in a small percentage of cases. Even though it is the consensus of medical opinion that there is no scientific foundation for the now popular vaccine treatment of colds, and that the occasional good results are due to coincidence, the vaccine treatment is worth trying.

The chlorine treatment of colds was frequently found to be helpful in simple, uncomplicated cases, when used in the first stage. When tonsils or sinuses were involved, this treatment had little effect.

Do not return to work until your cold is over. If the cold hangs on after two weeks, or if there is any question as to whether it is an ordinary cold or not, consult your family doctor.

Last week, Dr. Diehl of Minneapolis reported having been successful in treating colds by using morphine, codeine, papaverine, and other opium extracts. He claims to have cut in half the time that a cold lasts and to abort it completely in some cases. This method of treatment will need further investigation before universal adoption and may prove to be very valuable. Like vaccines, chlorine, and so forth, it will have to be used under the supervision of your family doctor.

CONCLUSION

The common cold is being recognized more and more as exacting a terrific toll in suffering and money each year, and as the cause of many more serious complications and diseases.

Many researchers have attacked the cold problem, but, it must be regretfully confessed, so far without any results.

The specific cause of a common cold is not known, but it is thought to be infectious, due to a filterable virus.

The absolute prevention on scientific grounds is not possible at present.

The treatment as outlined is designed to prevent infection, to abort the cold if possible, to relieve suffering, to shorten the length of time that a cold lasts, and to prevent the occurrence of complications.

From the outline presented, it is apparent that there is no simple or specific treatment of the common cold, unless it be the dictum: "Stay in bed!" for staying in bed will not only cure your own cold, but will also prevent your spreading it to others.

There is an old cynical medical joke which will still hold good until a great deal more is known about the cause and treatment of the common cold, and that is, that "an untreated cold lasts fourteen days, while a treated cold can be cured in two weeks."

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PUBLIC HEALTH NOTES

J. ROSSLYN EARP, DR. P. H.
Director New Mexico State Bureau of Public Health.

DIABETICS STILL DIE

That such a revolutionary discovery in medicine as the discovery of insulin should have made so little impression on the death rate of that disease for which it is specific, is to me a never-failing source of astonishment. The easy explanation is that insulin is not a cure for diabetes. This does not impress me. Insulin is capable of preventing death from diabetes and if death from diabetes is postponed long enough the patient will surely die of something else. It is true that, when diabetes is recorded on a death certificate as one out of two or more causes of death, the statisticians will classify the death as due to diabetes (since they must choose **one** cause for statistical purposes) in preference to such causes as influenza, measles, dysentery, rheumatic fever, diseases of the heart, the pneumonias, and some others. But there remain 127 ways is which the diabetic can die without any chance of diabetes taking the statistical blame. Note, for example, that if a patient dies from diabetes and tuberculosis, tuberculosis takes precedence as the cause of death.

A more plausible excuse is given by Dublin[1] when he says: "The use of insulin has served to increase the number of cases after age 45, and in this way has helped to shift the mortality from the younger ages to the older ones." This does in fact explain that, while the death rate from diabetes has been increasing at ages over 45, it has been decreasing at ages under 45. But it does not explain away the distressing fact that at all ages (taken together) the death rate of diabetes is still steadily increasing. A generation ago diabetes was twenty-first among the causes of death; today it stands tenth.

Then there is the explanation offered by Bolduan[2] "that the increase, during the past 30 or 40 years, in the diabetes mortality in the various age groups, finds an adequate explanation in the more frequent recognition of the disease in recent years." Is this the answer? Has diabetes always been tenth among the causes of death and we have only just found it out? It has been possible to detect sugar in human urine since 1842, and Fehling's quantitative test for sugar has been available since 1848. Yet, says Doctor Bolduan, routine urine analysis for sugar was not introduced into the New York Hospital until some time between 1880 and 1890; and, he adds, "undoubtedly the general practitioner lagged far behind." Perhaps Anyhow the general public lags far behind the general practitioner. In New Mexico we have a relatively low death rate from diabetes. But here no less than one-third of our deaths take place without medical attention. When this third of the population receives medical attention there can be no doubt that the number of diabetes deaths will appear to increase.

But when all is said and done, it is still hard for me to avoid the conviction that since 1921, when Banting discovered insulin, a lot of people have died from diabetes who ought to have died from something else.

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COMING MEETINGS

Of particular interest, and immediately ahead of us, is the Southwestern District Conference of the American College of Surgeons, which will be held in Phoenix on March 16 and 17. Complete program will be found elsewhere. Partly because the meeting will immediately follow the much larger California conference, an unusually large and eminent group of surgeons will be at the Phoenix conference. Surgeons from over the Southwest will certainly not wish to miss this meeting and program. The medical profession of the Southwest have been trying for years to establish that district as an entity; for this purpose the Medical & Surgical Association of the Southwest was conceived. When the district conferences of the College of Surgeons were started, effort was made to enlist their cooperation in consolidating and welding together the medical profession of western Texas, New Mexico and Arizona. The College has been feeling its way carefully in this matter, first including El Paso with the balance of Texas, then trying out New Mexico and Colorado, and this year they proposed to include Arizona with California. However some of the Fellows of the College in Arizona objected to this, and, with Dr. Franklin Martin's influence to support them, the forthcoming District Conference was planned. Should this meeting prove an outstanding success, it will go far toward establishing the "Southwest District" as a geographical and professional entity. It is, therefore, important that it be supported enthusiastically.

The New Mexico meeting will be held in Las Vegas during May, the exact dates not being known to this office at the time of going to press.

The Arizona state meeting will be in Prescott early in June, and plans are already under way for an outstanding program. It will be very agreeable to have this meeting somewhat later, immediately preceding the national meeting.

The American Medical Association will be held in Cleveland in June, with a week's interval between it and the Arizona meeting.

Watch for full details of these meetings in later issues of this journal.

PROGRAM

Friday, March 16, 1934

REGISTRATION AND INFORMATION—
LOBBY, HOTEL WESTWARD HO

CLINICS
9:00—12:00 a. m.
Good Samaritan Hospital
St. Joseph's Hospital

HOSPITAL CONFERENCE
Main Dining Room, Hotel Westward Ho
9:00—12:00 a. m.

Round Table Conference and Discussion of Administrative, Professional, and Economic Problems: Conducted by Robert Jolly, Houston; Superintendent, Memorial Hospital, Malcolm T. MacEachern M. D., Chicago; Associate Director American College of Surgeons, and Director of Hospital Activities.

MEDICAL MOTION PICTURES
Main Dining Room, Hotel Westward Ho
12:30—2:00 P. M.

Presented by Malcolm T. MacEachren, M. D., Chicago.

HOSPITAL CONFERENCE
Main Dining Room, Hotel Westward Ho
2:30—5:00 P. M.

Administrative, Professional, and Economic Problems of the Members of the Medical Profession, Trustees, Hospital Executives, and Others.

MEETING OF FELLOWS
Main Dining Room, Hotel Westward Ho
5:00—5:30 P. M.

Business Meeting of Fellows of the American College of Surgeons—Election of State Executive Committees for Arizona, and New Mexico.

COMMUNITY HEALTH MEETING
Phoenix Union High School
8:00 P. M.

Winfred Wylie, M. D., Phoenix, Chairman, Arizona State Executive Committee, Presiding.

THE AMERICAN COLLEGE OF SURGEONS—ITS MESSAGE TO YOU:
Franklin H. Martin, M. D., Chicago, Director General, American College of Surgeons.

CANCER—IT IS A CURABLE DISEASE:
Robert B. Greenough, M. D., Boston, President-Elect, American College of Surgeons.

SCIENTIFIC MEDICINE—ITS HISTORY:
Howard C. Naffziger, M. D., San Francisco; Professor of Surgery, University of California Medical School.

PROGRESS OF MEDICAL SCIENCE:
Allen B. Kanavel, M. D., Chicago; Professor of Surgery, Northwestern University Medical School.

SAVING YOUR EYESIGHT:
Edward H. Cary, M. D., Dallas; Professor of Ophthalmology and Otolarynology, Baylor University College of Medicine.

PERIODIC PHYSICAL EXAMINATIONS:
Thomas M. Joyce, M. D., Portland; Associate Clinical Professor of Surgery, University of Oregon Medical School.

SINUSITIS—ITS CAUSES AND PREVENTION:
Gordon B. New, M. D., Rochester; Professor of Rhinology, Larygnology, and Stomatology, Mayo Foundation.

ENCOURAGEMENT OF CANCER PATIENT:
Charles A. Dukes, M. D., Oakland; Vice-President-Elect, American College of Surgeons.

DOCTORS, PATIENTS, AND HOSPITALS:
Robert Jolly, Houston; President-Elect, American Hospital Association.

GOOD HOSPITAL CARE—Talking Motion Picture:
Presented by Malcolm T. MacEachern, M.D., Chicago; Associate Director, American College of Surgeons.

Saturday, March 17

CLINICS
9:00—12:00 A. M.
Good Samaritan Hospital
St. Joseph's Hospital
10:00—12:00 A. M.

CANCER CLINIC
St. Joseph's Hospital
Robert B. Greenough, M.D., Boston.

HOSPITAL CONFERENCE
Assembly Room, Good Samaritan Hospital
9:00—12:00 A. M.

Demonstrations and Round Table Conference dealing with departmental activities and various phases of Hospital Standardization and Administration: Conducted by Robert Jolly, Houston;

Convention Headquarters

For the Southwestern Sectional Meeting of
AMERICAN COLLEGE OF SURGEONS
Phoenix, March 16, 17

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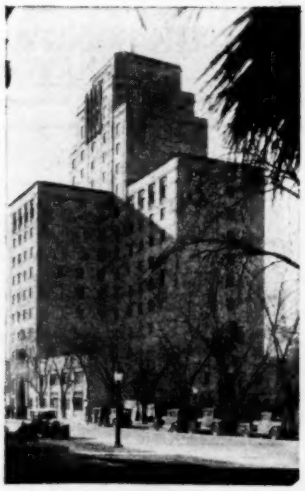


and Malcolm T. MacEachern, M. D., Chicago; assisted by Mrs. J. O. Sexson, Superintendent, and heads of departments.

MEDICAL MOTION PICTURES
Main Dining Room, Hotel Westward Ho
12:30—2:00 P. M.

Presented by Malcolm T. MacEachern, M. D., Chicago.

SCIENTIFIC MEETING
Main Dining Room, Hotel Westward Ho

Treatment of Tuberculosis of Tendon Sheaths:
Allen B. Kanavel, M. D., Chicago; Professor of Surgery, Northwestern Univerty Medical School.

Topic speaker to be announced later.

Results in the Treatment of Carcinoma of the Breast:
Thomas M. Joyce, M. D., Portland; Associate Clinical Professor of Surgery, University of Oregon Medical School.

Topic speaker to be announced later.

The Cervical Rib Syndrone with and without Cervical Ribs:
Howard C. Naffziger, M. D., San Francisco; Professor of Surgery, University of California Medi-School.

Topic speaker to be announced later.

SCIENTIFIC MEETING
EYE, EAR, NOSE AND THROAT
Conference Room A, Mezzanine Floor
Hotel Westward Ho
2:30—5:30 P. M.

Ocular Manifestations of Nasal Origin:
Edward H. Cary, M. D., Dallas; Professor of Ophthalmology and Otolaryngology, Baylor University College of Medicine.

Concerning a Study of Retinal Vascular System:
D. F. Harbridge, M. D., Phoenix.

Tumors of the Pharynx and Larynx:
Gordon B. New, M. D., Rochester; Professor of Rhinology, Laryngology, and Stomatology, Mayo Foundation.

Topic speaker to be announced later.

HOSPITAL CONFERENCE
Nurses' Class Room
St. Joseph's Hospital
2:00—5:00 P. M.

Demonstrations and Round Table Conference dealing with departmental activities and various phases of Hospital Standardization and Administration: Conducted by Robert Jolly, Houston; and Malcolm T. MacEachern, M. D., Chicago; assisted by Sister M. Monica, Superintendent, and heads of departments.

SCIENTIFIC MEETING
Main Dining Room, Hotel Westward Ho
8:00—10:00 P. M.

Treatment of Cancer of the Breast:
Robert B. Greenough, M. D., Boston; Consulting Surgeon, Massachusetts General and Collis P. Hunting Memorial Hospitals.

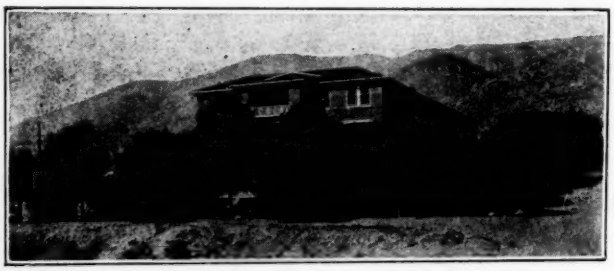
Small Intestinal Tumors:
Thomas M. Joyce, M. D., Portland.

Practical Considerations in the Prevention of Defects in the Skull and Their Repair:
Howard C. Naffziger, M. D., San Francisco.

Teleplasms Within and Around the Eye:
Edward H. Cary, M. D., Dallas.

Current Problems in Reconstructive Surgery of the Face:
Gordon B. New, M. D., Rochester.

Chest Surgery and the General Surgeon:
Charles A. Dukes, M. D., Oakland; Chief Surgeon, Alameda County Hospitals, West Division.



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THE WOMAN'S AUXILIARY TO THE ARIZONA STATE MEDICAL ASSOCIATION

MRS. J. C. WILSON
Willcox, Ariz.

The National President's letter in the December issue of the A. M. A. Bulletin overflowed with items of interest to every Auxiliary member and friend. Mention of the splendid activity of the Idaho State Auxiliary and the Oregon Auxiliary is made and in giving outline of the annual meeting of the National Auxiliary in Cleveland, Ohio, in June, the President laments the fact that there is no state auxiliary to work with. She brings to our attention the porgram of work under the direction of Dr. Martha Elliot, director of the Division of Child and Maternal Health, Children's Bureau, United States Department of Labor, who is traveling from state to state in the interest of the child health recovery program. This, as well as many Federal plans, should be studied by the Auxiliary so that our knowledge of them may be first hand. Our attention is called to the article on amebic dysentery which was published in the Journal of the A. M. A. of November 18th, and we are advised to read it and be ready to talk intelligently on that subject.

When we read that in Arizona, alone, deaths from automobile accidents during the month of December numbered 21, it should bring to us the outstanding need for some active program of safety.

The Pima County Auxiliary held its annual meeting recently and elected officers and heard reports of the officers telling of the work of 1933. This was a real fine meeting with much interest.

We sympathize with Dr. John Hagan of Miami, in the death of his wife, Julia Purcell Hagan, a native of Arizona.

It is hoped that next month will have more news and the Press Chairman asks you to please let her have information regarding your accomplishments.

Since writing the above another News Letter has been received from our national officers. Speaking of membership, our President, Mrs. Blake, says: "To have a highly successful meeting (national convention) we must needs have delegates in large numbers, and to accomplish this there are several things to be done. First, we need to reinstate our delinquents. We need them and we are equally certain they need us. Second, every eligible woman who has not yet joined should be given a chance to know the delightful social experiences and the important opportunities, in becoming a unit in the ranks of this great reserve army, that she is missing."

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Holden and Gurnee: Am. J. Obs. & Gyn., Vol. XXII, No. 1, p. 87, July, 1931.

Graham: Elliott Treatment of Pelvic Inflammation, Am. J. Surg., 16: 423-426, June, 1932.

Counsellor: Treatment of Chronic Infection of the Pelvis: A Consideration of the Elliott Method, J.A.M.A., p. 916, September 16, 1933.

Stone: Am. J. Phys. Ther., April, 1932.

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Central and Southern Zone	Northern Zone
Cottonwood—Feb.	Cottonwood—March.
Ash—late Feb., March.	Junipers—March.
Rabbit Bushes—Mar., Apr.	Box Elder—April.
Bermuda Grass — March, Nov.	Scrub Oak—May, June.
Johnson Grass — March Nov., relieved by Bermuda ext.	Russian Thistle — June, Aug.
Careless Weed—May, Oct.	Careless Weed—June., Sep.
Russian Thistle—May, Jul.	Western and Bur Ragweeds Aug., Sept.
Olive—May.	Slender Ragweed in some sections—Aug., Sept.
Slender Ragweed — Sept. Nov.	

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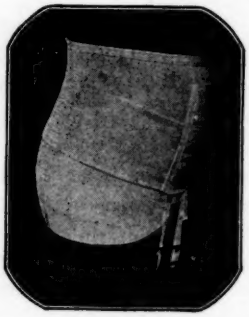

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